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ANATOXIN AS AN IMMUNIZING AGENT AGAINST DIPHTHERIA *

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AND
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PARIS, FRANCE

It is with much interest that we have read in the AMERICAN JOURNAL OF DISEASES OF CHILDREN the work on "Comparative Effects of Diphtheria Toxoid and Toxin-Antitoxin as Immunizing Agents," by Dr. G. F. Weinfeld and Dr. M. Cooperstock.[1] In a series of publications, and especially in our article on diphtheria prophylaxis in France,[2] we have already made known the methods of vaccination by toxoid and showed its use in prophylaxis.

In a recent study, G. F. and G. H. Dick [3] affirmed the superior value of anatoxin, or toxoid, over toxin-antitoxin. They said: "Diphtheria toxoid as prepared by Ramon and given in the three doses recommended by him, is a better immunizing agent than O I L + diphtheria toxin-antitoxin mixtures, even when five doses of the latter are given."

The work of Dr. Weinfeld and Dr. Cooperstock confirms this superiority, and affirms: "the necessity of the use of toxoid in preference to any other immunizing agent in the prophylaxis of diphtheria."

Our object in this short article is to indicate what has been done in France in the past few months with regard to the question of immunization by toxoid.

The recommendation of the French Academy of Medicine, as well as the help of the Minister of Hygiene, who has given instructions for generalizing the use of toxoid, and also the keenness of the various medical schools and medical staffs, has been a great incentive during the past months for popularizing this form of vaccination among the

* Submitted for publication, Nov. 6, 1929.

* From the Institut Pasteur and the American Hospital.

1. Weinfeld, G. F., and Cooperstock, M.: Comparative Effects of Diphtheria Toxoid and Toxin-Antitoxin as Immunizing Agents, Am. J. Dis. Child. **38**:35 (July) 1929.

2. Ramon, G., and Hélie, G. I.: Diphtheria Prophylaxis in France: Experiments with Anatoxin Vaccination, J. A. M. A. **91**:1028 (Oct. 6) 1928.

3. Dick, G. F., and Dick, G. H.: Comparative Value of Toxoid and Toxin-Antitoxin Mixtures, J. A. M. A. **92**:1901 (June 8) 1929.

French people. In 1927, the number of vaccinations was 50,000; in 1928, it had reached 300,000, and during the first six months of 1929 it was 500,000.

In Paris and in a great number of cities vaccination is carried out in an organized manner, not only in hospitals but in families, parents' associations, children's welfare centers, consultations for children, schools, etc. The standard technic is still the same, namely: an injection of 0.5 cc.; after three weeks, a second injection of 1 cc., and two weeks later, a third injection of 1.5 cc.

By using toxoid prepared at the Institut Pasteur by one of us, toxoid of which the intrinsic antigen value is proved by the flocculent reaction and which is never inferior to 10 antigenic units, we obtain in from six weeks to two months a negative Schick reaction in from 96 to 100 per cent of the cases that showed a positive reaction before vaccination. This immunity is lasting. It has been found that in cases of three or four years' duration only from 1 to 2 per cent showed reactions to the Schick test. In the serum of the great majority of the cases from $\frac{1}{10}$ to $\frac{1}{3}$ or even more than 1 unit of antitoxin was seen, which represents three, ten or thirty times the amount necessary ($\frac{1}{30}$ of a unit) to cause a negative Schick reaction.

Only the future will show the real influence of toxoid vaccination on diphtheria morbidity, but the results obtained in certain centers where toxoid vaccination has been carried out for several years give interesting information. For instance, the 1,200 inmates of the Hôpital Maritime de Berck, a hospital for patients with tuberculosis of the bone, the following results were obtained when antidiphtheritic serum was used: in 1923, four deaths in thirty-four cases; in 1924, three deaths in thirty-six cases; in 1925, five deaths in sixty-seven cases. In 1926, toxoid vaccination was introduced and practiced on a great many patients at this hospital, and in 1927 and 1928 it was practiced on all new patients. In 1928, there were only two very slight cases of diphtheria.

M. and G. Mozer [4] stated in 1928 that diphtheria has practically disappeared from the Hôpital Maritime, where diphtheria constantly occurred and where there were severe cases before the patients were vaccinated. It is interesting that even these tuberculous patients give slight reactions to toxoid.

This example shows the real efficiency of anatoxin against diphtheria; if such results can be obtained in one place, they should be obtained throughout a country. For this purpose, vaccination of infants should be systematically adopted; only if this is done will the number of cases of diphtheria gradually decrease and eventually disappear from all countries.

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4. Mozer, M.; Mozer, G., and Cofino, E.: Bull. et mém. Soc. méd. d. hôp. de Paris **52**:1443 (Oct.) 1929.

COMPARATIVE STUDIES OF THE INFLUENCE OF ACID-FORMING AND BASE-FORMING DIETS ON THE METABOLISM OF RATS *

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AND
I. NEWTON KUGELMASS, M.D.
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Infant feeding regimens formulated by pediatricians of representative institutions have been calculated by us to be acid-forming; that is, the dietaries contain an excess of acid-forming elements (chlorine, sulphur and phosphorus) over basic elements (sodium, potassium, calcium, magnesium and iron) (table 1). Prolonged milk mixtures [1] used in the feeding of very young infants, however, are all base-forming. The daily requirements of inorganic elements during growth indicate a preponderance of basic over acid-forming elements [2] (table 2). This is actually reflected in the inorganic composition of the child's body, (table 3).

The growing young animal requires these inorganic constituents—acid-forming and base-forming—for the maintenance of a labile acid-base mechanism, for the normal contractility of the muscular system, the normal irritability of the nervous system, for the physiologic osmotic pressure relations, for the establishment of membrane equilibria (Donnan), acids and alkalis of digestive secretions, for the synthesis of tissues (bone, blood, thyroid), for the maintenance in solution of metabolites (uric acid), etc.

These specific functions require qualitatively the presence of acid and basic elements. The correlations of all these functions becomes manifest in the living organism by the equilibrium of physicochemical mechanisms involving the inorganic ions.[3] The maintenance of such an equilibrium (health) requires the presence of inorganic elements in all tissues in quantitative amounts.

The lack of accord between the practices in infant feeding and the metabolic principles indicating the inorganic requirements of the growing infant has led to the present investigation. The effects of natural acid-

* Submitted for publication, Oct. 30, 1929.

* From the Department of Pediatrics, the Fifth Avenue Hospital.

1. Powers, Grover: Comparison and Interpretations on Caloric Basis of Milk Mixtures Used in Infant Feeding, Am. J. Dis. Child. **30**:453 (Oct.) 1925.

2. Shohl, A. T.: Physiol. Rev. **3**:509, 1923.

3. Kugelmass, I. N.: J. Biol. Chem. **58**:649, 1924.

TABLE 1.—*Diet for 6-Month Infant (Calories 750); Fifth Avenue Hospital*

Time	Food	Amount	Acid	Base
6 a. m.	Milk	6 oz.	....	4.2
8 a. m.	Orange juice	2 oz.	....	3.3
10 a. m.	Cereal	4 oz.	14.1	...
	Milk	6 oz.	....	4.2
2 p. m.	Milk	6 oz.	....	4.2
	Egg yolk	1 oz.	6.6	...
	Vegetable	2 oz.	....	3.2
6 p. m.	Cereal	4 oz.	14.1	...
	Milk	6 oz.	....	4.2
	Acid over base value, 11.5		34.8	23.3

Diet for 2-Year Child (Calories 1,500); Johns Hopkins Hospital

8 a. m.	Cereal	3 oz.	11.5	...
	Sugar	2 tsp.	....	...
	Milk	6 oz.	....	4.2
	Toast with butter	1 oz.	3.4	...
	Egg	1	6.6	...
12:30 p. m.	Soup	3 oz.	....	...
	Toast with butter	¾ oz.	2.6	...
	Scraped beef	2 oz.	7.1	...
	Potato or	2 oz.	....	0.3
	Rice	2 oz.	0.4	...
	Green vegetable	2 oz.	....	3.2
	Dessert (custard)	1½ oz.	0.2	...
	Milk	6 oz.	....	4.2
6 p. m.	Egg	1	6.6	...
	Toast with butter	1 oz.	3.4	...
	Stewed fruit	2½ oz.	....	2.8
	Milk or cocoa	6 oz.	....	4.2
	Acid over base value, 22.9		41.8	18.9

TABLE 2.—*Daily Mineral Requirement in Childhood*

Basic		Acidic	
Element	Gm.	Element	Gm.
Calcium	1.0	Phosphorus	1.5
Magnesium	0.8*	Sulphur	0.4*
Iron	0.015	Chloride	0.8*
Sodium	0.35*		
Potassium	1.37*		
Base	3.53	Acid	2.7

Balance: 0.8 Gm. base

* Approximated.

TABLE 3.—*Retention of Mineral Salts by New-Born Infant per Gram of Ash*

Basic	Gm.	Cc. 0.1 N	Acidic	Gm.	Cc. 0.1 N
Calcium	0.28	140	Phosphorus	0.16	96
Magnesium	0.009	7	Sulphur	0.008	5
Sodium	0.05	25	Chloride	0.06	18
Potassium	0.05	15			
Bases		186	Acids		119

Balance: 67 Cc. 0.1 N base

forming and base-forming diets on the metabolism of young rats have been studied from the physical, chemical and pathologic standpoints.

LITERATURE

Variations in the acid-base value of the diet have been shown to influence the absorption and excretion of calcium and phosphorus in rickets and tetany.

Bartlett and his associates [4] found that the addition of vegetables (base-forming) favorably influenced the metabolism of young infants.

Zucker [5] made a rickets-producing diet antirachitic by decreasing the acid-forming constituents.

McClendon,[6] on the other hand, increased the rickets-producing value by the addition of alkali.

Jones [7] cured a patient with rickets by the addition of acid to the diet.

Shelling,[8] in Park's Laboratory, found that acid diets produce a more severe degree of rickets than do alkaline diets.

Chaney and Blunt [9] found that orange juice added to the diet markedly increased the retention of calcium by an amount considerably greater than that contained in the orange juice.

Eddy [10] found that bananas added to the diet have the same marked effect on the retention of calcium.

Binger [11] produced tetany in dogs by the intravenous injection of alkaline phosphate, and his results were confirmed by Salvesen, Hastings and McIntosh.[12]

Rinjiro [13] observed the maximal growth of rats on rations highest in mineral content, and Randoin [14] confirmed this observation, especially with the salts of the alkaline metals.

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4. Bartlett, F. H.; Courtney, A. M., and Fales, H. L.: Some Analyses of Vegetables Showing the Effect of the Method of Cooking, Am. J. Dis. Child. **14**:34 (July) 1917.

5. Zucker, T. F., and Johnson, W. C.: Proc. Soc. Exper. Biol. & Med. **20**:20, 1922.

6. McClendon, J. F.: Am. J. Physiol. **74**:694, 1927.

7. Jones, M. R.: Am. J. Physiol. **74**:694, 1927.

8. Shelling, D. R.: The Production of Rickets in Rats by Changes in Acid-Base Ratio of the Diet, Thesis, Yale University, 1925.

9. Chaney, M. S., and Blunt, K.: J. Biol. Chem. **66**:829, 1925.

10. Eddy: Personal communication to the authors.

11. Binger, C.: J. Pharmacol. & Exper. Therap. **10**:105, 1917.

12. Salvesen, H. A.; Hastings, A. B., and McIntosh, J.: J. Biol. Chem. **60**:311, 1924.

13. Rinjiro S.: J. Sc. Agric. Soc., Japan, 1924, no. 256, p. 138.

14. Randoin, L., and Alquier, J.: **180**:2063, 1925.

Page [15] found that the alkaline reserve of the blood of rabbits fed on acid-forming diets was lower than the blood of those fed on base-forming diets.

Borak [16] deduced that dogs on acidic diets gain in weight and have a high water retention and a lowered nitrogen retention in comparison with those on basic diets.

Steenbock and his associates [17] have shown that the utilization of iron is favored by the addition of cabbage.

EXPERIMENTAL

Comparative studies of acid-forming and base-forming diets were made on two groups of twenty-three and twenty-five young albino rats weighing between 30 and 35 Gm. Litters of the same stock and age were maintained on natural foods—acid and base predominating in composition—in light and in darkness during all the seasons. The composition of the acid-forming and the base-forming diets is given in table 4.

TABLE 4.—*Acid-Forming Diet*

Food	Weight, Gm.	Protein	Carbohydrate	Fat	Calories	Acid	Basic	Vitamin
Egg	45 Gm.	6.1	....	4.7	68.7	4.95		ABDE-G A-P
Rice	60 Gm.	4.8	47.4	0.2	215.9	5.0	....	.....
Oatmeal	40 Gm.	6.7	26.5	2.9	163.1	5.1	....	ABE
Agar	2 Gm.	....	....	...	.....	....	....	.....
		17.6	73.9	7.8	447.7	15.05	....	ABDE A-P
		Base-Forming Diet						
Klim	20 Gm.	5.2	7.6	5.0	105.0	....	3.3	ABCDE
Potato	100 Gm.	2.5	20.9	0.1	96.9	....	7.7	ABC
Beans (lima)	60 Gm.	10.8	39.6	0.9	210.0	....	24.0	ABE
Dextrin	3 Gm.	....	2.7	...	17.0	....	....	.....
Agar	2 Gm.	....	....	...	.....	....	....	.....
		18.5	70.8	6.0	428.9	....	35.0	ABCDE

The cereals of the acid-forming diet were first cooked over the direct flame for a few minutes and then placed in a steamer where the cooking process continued for several hours, whereas the lima beans and potatoes of the base-forming diet were cooked only in the steamer. The other ingredients were added later when the cooked material had cooled. These mixtures were prepared on the average of three times a week and were kept in refrigeration between from 40 to 45 F.

METHODS

Total base determinations were made by a modified method of van Slyke, Wu and McLean.[18] The calcium determinations were made by

15. Page, I. H.: Am. J. Physiol. **66**:1, 1923.

16. Borak, J.: Biochem. Ztschr. **135**:480, 1923.

17. Steenbock, H., and others: J. Biol. Chem. **66**:425, 1925.

18. Van Slyke, D. D.; Wu, Hsien; and McLean, F. C.: J. Biol. Chem. **56**: 818, 1923.

Kramer and Tisdall's modification. The phosphate determinations were made according to Briggs' method.[19] The carbon dioxide and chloride contents were determined according to the methods of van Slyke and Neill.[20] The p_H determinations were made according to the method of Hastings and Sendroy.[21] The dextrose was determined according to the method of Lewis and Benedict. The cholesterol was determined by the method of Bloor and Knudson.[22] The albumin and globulin were determined by Howe's modification.[23] Serum protein was determined refractometrically. The bone value was estimated by the method of Chick and of Bethke, Steenbock and Nelson.[24]

RESULTS

Analyses of the Blood.—The average values at the end of twenty-eight weeks, obtained on the series of animals reared on acid-forming

TABLE 5.—*The Chemical Composition of the Blood of Rats on Acid and Basic Diets*

	Inorganic						Organic							
Diet	Carbon Dioxide	p_H	Chlorides	Phosphorus	Calcium	Total Base	Sugar	Cholesterol	Protein	Fat I No.	nD	Clotting Time	Hemoglobin	Red Blood Cells
Neutral.....	48	7.27	280	6.1	9.0	148	135	140	6.5	58	1.347	2.5	113	9.5
Basic: Sunlight....	50	7.48	261	7.5	9.8	154	175	110	6.7	62	1.347	2.0	110	8.8
Darkness...	51	7.38	282	6.3	8.2	143	160	130	6.6	..	1.342	3.0	...	....
Acid: Sunlight.....	48	7.28	305	5.7	11.8	137	150	150	6.9	51	1.345	3.0	105	10.9
Darkness....	44	7.23	317	6.0	11.3	134	144	142	7.1	..	1.344	2.0	...	....
Base-acid.....	51	7.33	353	3.8	10.0	142	140	120	6.7	55	1.347	3.5	110	10.2
Acid-base.....	49	7.38	320	4.6	9.7	150	145	137	6.7	63	1.345	2.5	102	8.6

and base-forming diets, are shown in table 5. They indicate that at the end of the experiment, when equilibrium on the diet had been established, the acidity or alkalinity of the diet had a marked effect on the animals.

The acid-forming diet depressed the serum phosphorus, the alkali reserve total and the p_H with a compensatory rise in the chloride, sugar and cholesterol. The base-forming diet produced the converse changes, with some depression in the serum calcium. The albumin-globulin

19. Briggs, A. P.: J. Biol. Chem. **53**:13, 1922.

20. Van Slyke, D. D., and Neill, J. M.: J. Biol. Chem. **61**:523 (Sept.) 1924.

21. Hastings, A. B., and Sendroy, J., Jr.: J. Biol. Chem. **61**:695 (Oct.) 1924.

22. Bloor, W. R., and Knudson, Arthur: J. Biol. Chem. **29**:7, 1917.

23. Howe, P. E.: J. Biol. Chem. **49**:109, 1921.

24. Bethke, R. M.; Steenbock, H., and Nelson, M. T.: J. Biol. Chem. **58**: 76, 1923.

ratios, percentage of fat and iodine content of the fat were all normal. The hematological data were also normal in both groups of animals.

Histologic Examination of the Bones.—The animals fed on acid-forming diets developed rickets manifested by thoracic deformities, enlargements of the costochondral junction and long bones, which were small, soft and brittle. Histologically, the bones showed considerable osteoid tissue with little lime deposition, wide and irregular metaphyses and swollen cartilage cells.

The rats fed on base-forming diets showed no thoracic deformities; the ephiphyses were somewhat enlarged and the extremities were large and firm. Histologically, the bones showed some semblance of rickets, a moderate amount of osteoid tissue with some lime deposition in the metaphyses and cartilage.

TABLE 6.—*Analyses of Femora of Rats*

Diet	Calcium Phosphorus Ratio	Age, Weeks	Weight Dry, Mg.	Weight Fat-Free, Mg.	Weight Ash, Mg.	Ash Fat-Free Fat, per Cent	Ash Fat-Free Bones, per Cent
Acid: sunlight; group I	0.062 : 294	28	375.4	326.0	178.8	13	54
Acid: sunlight; group II	0.057 : 361	28	213.4	181.8	89.2	15	50
Acid: darkness; group III	0.062 : 294	28	234.3	215.6	107.6	8	49
Acid: darkness; group IV	0.057 : 361	28	170.4	148.0	73.4	13	49
Base: sunlight; group V	0.080 : 278	28	315.6	304.2	186.0	3.6	61
Base: darkness; group VI	0.080 : 278	28	385.4	354.2	207.3	8	58
Analyses of Humeri of Rats							
Acid: sunlight; group I	0.062 : 294	28	178.4	160.6	71.6	10	55
Acid: sunlight; group II	0.057 : 361	28	99.4	90.3	45.0	9	50
Acid: darkness; group III	0.062 : 294	28	111.8	111.0	48.3	8	43
Acid: darkness; group IV	0.057 : 361	28	103.4	95.3	47.2	7	50
Base: sunlight; group V	0.080 : 278	28	168.6	159.4	118.2	5	61
Base: darkness; group VI	0.080 : 278	28	191.4	179.6	108.4	6	60

The histologic observations are in accord with the results previously recorded by Shelling [8] in Park's laboratory.

Analysis of the Bones.—Analyses of the humeri and femora are given in table 6. The percentage composition of the bones determined in this way offers a fundamental basis of comparison of the bones of animals reared on different diets.

The animals on the base-forming diet showed the greater percentage of ash and the heavier bones while those on the acid-forming diet showed the smaller percentage of ash and the lighter bones; the animals that had been exposed to light showed a higher ash content than those in darkness.

Metabolism.—The rats on base-forming diets showed a more marked average gain in weight than those on the acid-forming diets (fig. 1), paralleling the observations of Shelling.[8] The weight contrast was striking for the summer, less so for the spring and least for the

winter (figs. 2 to 7). Other metabolic properties which characterized each group of animals are summarized in table 7.

The bones of the animals on the basic diet were large, well formed, round and thick while those of the animals on the acid diet were small, thin and brittle. The analyses of the bones of the "acid" rat are given in table 6.

COMMENT

Biochemical Results.—The blood constituents involved in the acid-base mechanism differ in the "acid" and "base" diet groups of the animal. The total base is definitely higher in the animals on the basic diet than in those on the acid diet, with corresponding variations in carbon dioxide combining power or alkali reserve. The chloride in the group on an acid diet is higher than that in the group on a basic

TABLE 7.—*The Metabolic Properties of the Rat Groups on Acid and Base-Forming Diets*

	Acid (Twenty-Three Rats)	Basic (Twenty-Five Rats)
Weight: Summer...	Average gain 7 Gm. per week (first 14 weeks)	Average gain 17 Gm. per week (first 14 weeks)
Winter....	Average gain 8 Gm. per week (first 14 weeks)	Average gain 8 Gm. per week (first 14 weeks)
Spring.....	Average gain 11 Gm. per week (first 14 weeks)	Average gain 18 Gm. per week (first 14 weeks)
Skeleton.....	Bones small, brittle, rachitic (histologic)	Bones large, firm, nonrachitic (histologic)
Musculature.....	Flabby; rat posture poor	Tonicity good; posture characteristic
Fur.....	Shaggy; matty; growth patchy	Silky, soft; growth uniform
Activity.....	Mild	Vigorous; irritable
Appetite.....	Fair	Good
Stools.....	Small, hard	Large, soft
Infections.....	Rats susceptible early	Susceptible later
Reproduction.....	Delayed (26th week in spring group)	Normal (15th week)

diet. These results probably define the extreme upper and lower levels of normal in blood values. The significance of this range of values becomes apparent if blood determinations are correlated with the nature of the dietary.

The variations of these acid-base factors may be illustrated graphically (fig. 8). The "normal" column consists of total base, *B*, and the sum of the concentrations of calcium, magnesium, potassium and sodium in combination with the acids of the plasma. The bases are obviously distributed as fixed base bound to sulphate and chloride (strong acids) and to protein, organic acids, phosphate and bicarbonate (weak acids). The base bound to the weak acids is available for combination with any strong acid, carbonic acid being liberated, etc., in accordance with the known buffering mechanism.

Rats on the acidic diet show a lowered alkali reserve and a compensated increase in chloride, while those on the basic diet show the converse, i. e., an elevated alkali reserve and a correspondingly lowered

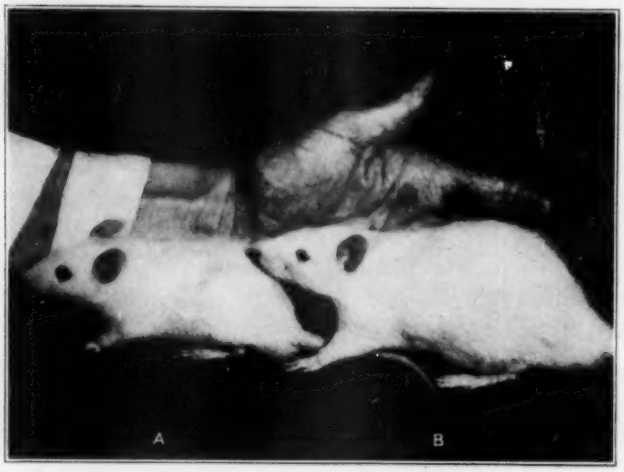

Fig. 1.—The rat on the left was fed on acid diet; the one on the right, a basic diet. The photograph shows the comparative size and appearance of typical rats of the same age and litter on acid and basic diets.

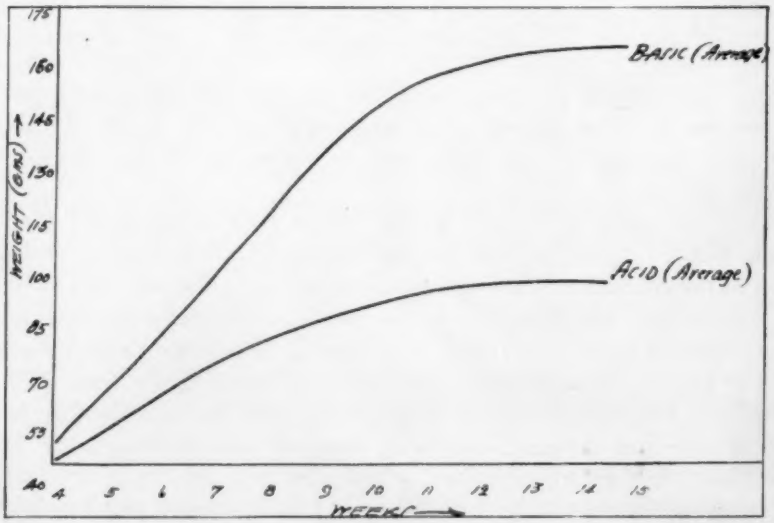

Fig. 2 (group I).—Summer. The weight curves of rats on acid and basic diets.

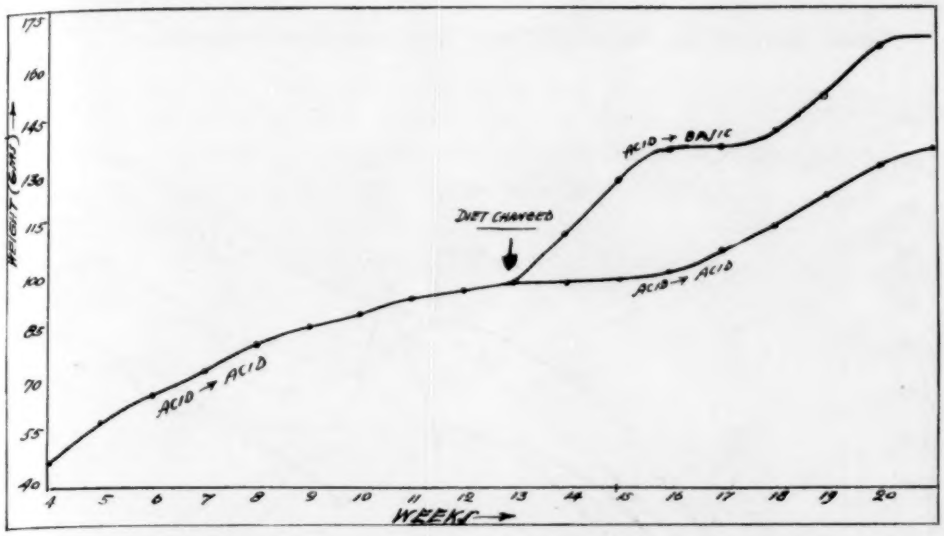

Fig. 3 (group I).—Summer. The weight curves of rats on an acid-forming diet.

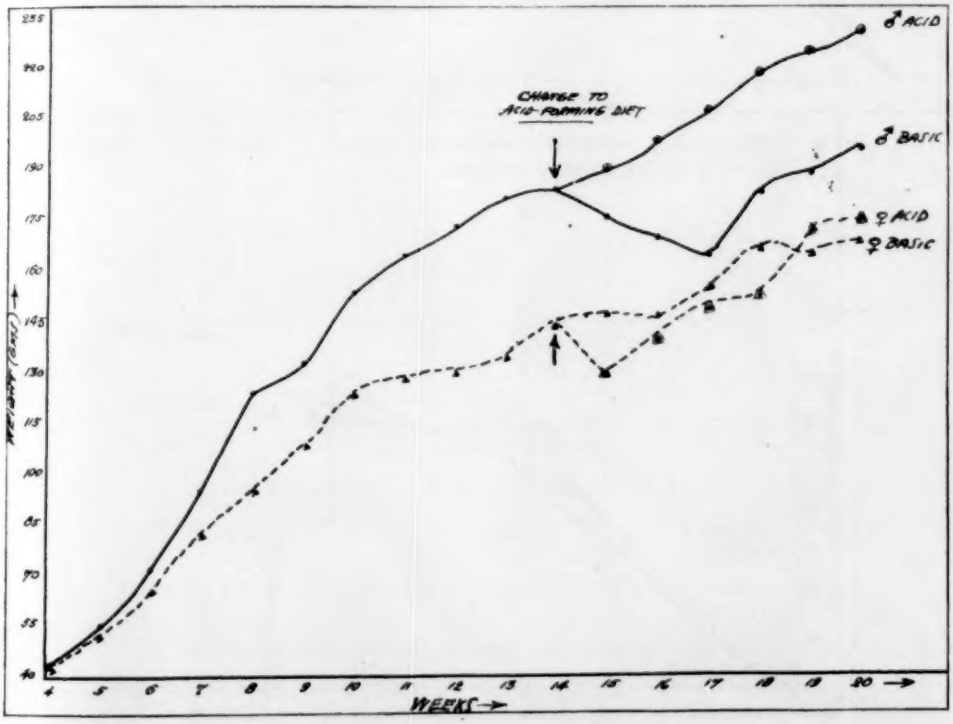

Fig. 4 (group I).—Summer. The weight curves of rats on base-forming diets.

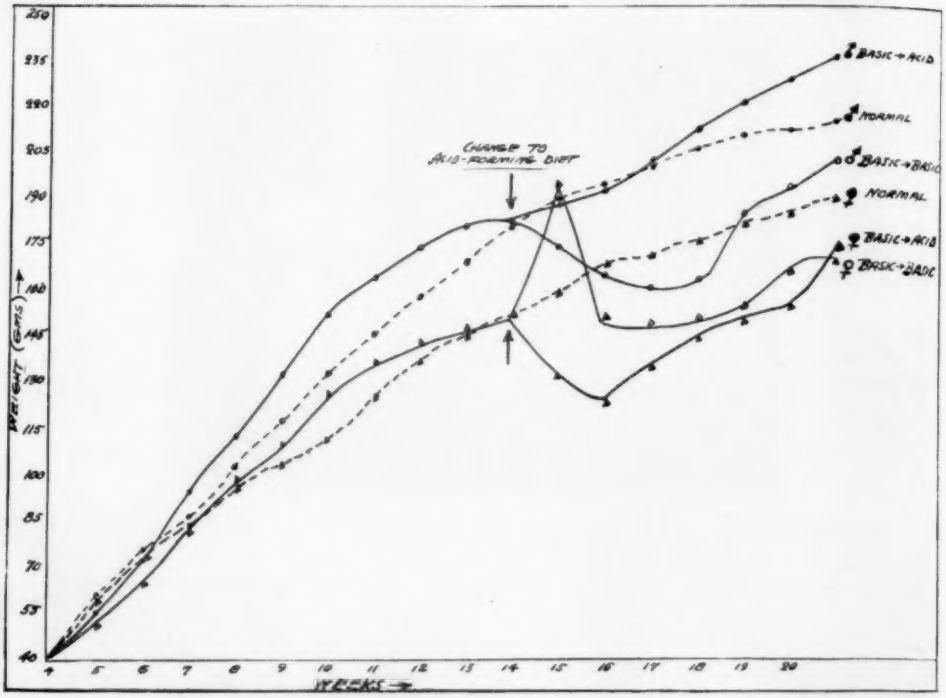

Fig. 5 (group I).—Summer. The weight curves of rats on base-forming diets; compared with the standard normals.

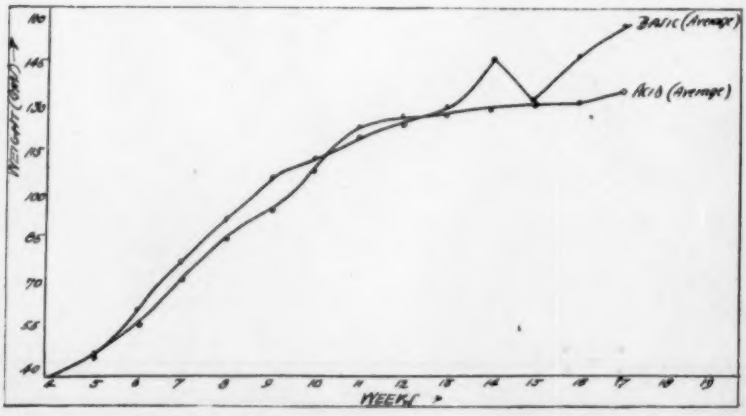

Fig. 6 (group III).—Spring. The weight curves of rats on acid and basic diets in darkness.

chloride level. The protein content in each group remained constant. The phosphate has been slightly higher in the "basic" animals. The p_H of the "basic" animals remained more on the alkaline side and that of the "acidic" animals more on the acid side of normal, 7.4.

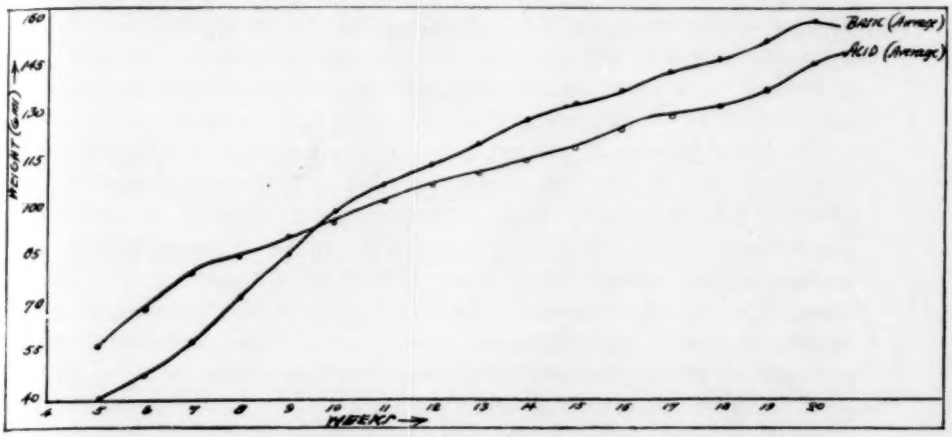

Fig. 7 (group II).—Winter. The weight curves of rats on acid and basic diets.

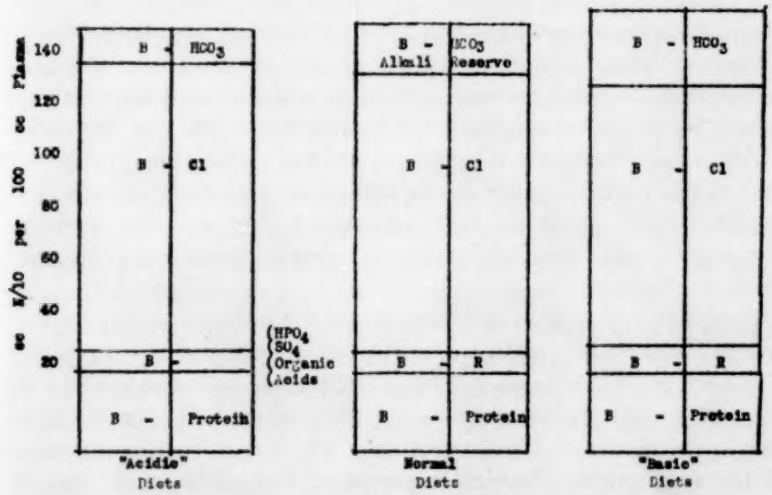

Fig. 8.—The acid-base distribution in the blood of rats.

The activity of the animal groups is a function of the biochemical status of the tissues—blood sugar, cholesterol, p_H, etc. The active "basic" animals have shown high blood sugar and occasional low cholesterol, while the inactive "acidic" animals have shown low blood sugars and suggestive high cholesterols. Glycolysis has already been associated with the alkaline state. Attempts to relate basal metabolism

of an animal in inverse ratio to the blood cholesterol have been found wanting.

The fat content and its degree of unsaturation in the animals on acid and basic diets are apparently analogous in each group. The iodine contents of the fats extracted from the animals in each group indicate the same combining power throughout. Evidently an alteration in the inorganic intake does not affect the lipin structure or content of a growing animal. The favorable effect of basic salts on calcification is unrelated to the kind of fat deposited in the body of the animal.

The blood plasma of rats on acid-forming and base-forming diets is evidently not of constant composition but is somewhat dependent, within the usually accepted range of normal, on the nature of the intake. This deviation in the constancy of composition of blood maintains when the food ingested contains the necessary elements at the minimum level of requirement. Living tissues have the property of selective assimilation—selecting necessary ingredients in suitable proportions and rejecting unsuitable or excessive ingredients provided the required elements are contained in the dietary in optimum amounts.

An illustration of this is afforded by Lansteiner's experiment. He fed young rabbits on meadow-hay exclusively for three months and another group of animals on cow's milk exclusively. These diets contained different relative amounts of sodium and potassium, hay being richer in potassium than in sodium, and milk richer in sodium than in potassium. Yet at the end of the period the composition of the blood obtained from the two groups, as regards sodium and potassium, was identical. This power of selection is effective when the substances available in the dietary are adequate in amount. Our experiments have subjected this selective power of the animal body to function with foodstuffs containing minimum requirements of the necessary elements. Under such a condition the power of selective assimilation is a less efficient mechanism.

Histopathologic studies of the bones of the animals on acid-forming and base-forming diets show a marked degree of rickets in the former and a very mild degree in the latter. Parallel determinations of the blood calcium and phosphorus of each group of animals revealed higher values in the rats reared on basic diets. These facts may be correlated with the fundamental chemical requirement for calcification—the concentrations of the ions, calcium, carbonate and phosphate must always exceed the solubility product of calcium phosphate and calcium carbonate, respectively, for precipitation of bone from blood of a normal rate:

$$[Ca^{++}]^3\ [PO_4^{=}]^2 > \text{Ks.p.}$$
$$[Ca^{++}]\ \ [CO_3^{=}] > \text{Ks.p.}$$

These ionic activities for bone formation are accelerated in a more alkaline medium (higher p_H and and higher carbon dioxide), facts

observed in the "basic" animals. This alkaline trend in the "basic" animals explains the high degree of irritability and activity of the animals in comparison with the acidic group.

Metabolic Results.—Base-forming foods promote increase in weight while acid-forming foods inhibit gain during growth. The cycle of events occurring during growth with a sufficiency of basic constituents in the dietary may be realized by a consideration of each of the factors involved in the processes of weight gain.

Weight = Σ (protoplasm, hydration, bone, etc.).

Protoplasm is electronegatively charged at the body reaction p_H 7.4 and necessarily requires for its maintenance in solution the electropositive basic ions for chemical combination. In fact, all cellular components are electronegatively charged and chemically combined with the basic ions.

Fe^{+++}	
Ca^{++}	Proteins
Mg^{++}	Organic acids
Na^{+}	Metabolites
K^{+}	

The requirements of basic ions which bind protoplasmic constituents become all the more striking since osmotic relations, membrane equilibria (Donnan), are dependent on ionized basic elements. In fact, the sum total of all protoplasmic components is a function of the base content of the tissues. The more adequately the basic ions are provided the greater will be the retention of all other required constituents. This retentivity is reflected in the gain in weight of growing animals on a sufficiency of basic elements in the dietary.

Evidence of the acquisition of increased protoplasmic mass on dietaries high in base value has been obtained from experiments on nitrogen balance. Asada [25] found a negative nitrogen balance or a retention of nitrogen constituents in dogs maintained on diets rich in basic salts.

Increased nitrogen retention involves a parallel rise in the water of hydration associated with the lipotropic colloid nature of proteins. Not only are the proteins chemically hydrated as protoplasm but also the basic ions are hydrated, the degree of hydration of electropositive ions (Ca^{++}, Mg^{++}, Na^{+}, K^{+}.) being considerably greater than that of the electronegative ions (Cl— $S^{=}P^{\equiv}$).

A sufficiency of intake of basic element accelerates metabolic activity and consequently also the growth increment. Asada found increased oxidization and better appetites in dogs on high basic diets. The inorganic basic constituents are therefore substrates of growth and regulators of cellular metabolism.

25. Asada, Kazuo: Biochem. Ztschr. **140**:327, 1923.

APPLICATION TO INFANT FEEDING

Rigid conclusions cannot be drawn from experiments on animals for infant feeding. The relation brought forward between the inorganic constituents in the diet and the metabolic state of a growing animal is, however, sufficient to warrant certain inferences significant in infant feeding.

Dilution of the mineral content of cow's milk (basic) by the addition of water and carbohydrates results in diminished intake of basic mineral matter in proportion to the energy-yielding constituents. The modern trend to concentrate feeding eliminates such possibility. In fact, the early offering of orange juice and cod liver oil favors the retention of basic elements.

During the period following weaning, there may be a mineral deficiency because of the practice of introducing into the infant's dietary refined cereal products—low in mineral content and acid-forming. This is corrected by supplementing the diet by fruit and vegetable juices or purees which have a marked positive base balance.

The first line of defense against rickets is the adjustment of the mineral content of the diet—a basic-forming diet favors the retention of calcium and phosphorus.

During the acute infections and gastro-intestinal disturbances associated with dehydration and depletion of basic salts, the addition of base-forming foods is corrective of the acidosis.

Throughout the period of growth, the animal requirement of a positive base balance of tenth-normal per kilogram of body weight is satisfied by daily inclusion in the dietary of adequate amounts of base-forming foods—milk, fruits and vegetables.

CONCLUSIONS

1. Acid-forming diets lacking in vitamin D produce rickets in young rats while base-forming diets under similar conditions produce a very mild degree of rickets.

2. Acid-forming diets shift the acid-base mechanism of young rats to the acid side and base-forming diets to the basic side of equilibrium.

3. Acid-forming diets inhibit and base-forming diets accelerate the rate of growth, development, metabolism and activity.

4. The base-forming food requirement during growth is considered in relation to infant feeding.

THE GROWTH AND NITROGEN METABOLISM OF INFANTS RECEIVING UNDILUTED MILK *

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For some time, ideas concerning growth have been undergoing a change. The rate of growth has been shown definitely to depend on the character and quantity of the food intake; consequently, as diets are improved, standards of growth become obsolete and are replaced by new ones. The present standards are based on averages of the growth of children who in all probability were not receiving an optimum diet. Chi Che Wang [1] observed that greater growth was obtained in children when the protein intake was approximately twice the accepted average requirement. The growth of a group of carefully supervised diabetic children [2] was found to exceed that which might be expected according to the usual standards for well children. Daniels [3] demonstrated that this principle is applicable also to infants. Though the infants observed by her received diluted milk, the food intake was greater than that administered for similar observations previously, and the growth of the infants was greater than that predicted by Finkelstein's formula,[4] a commonly used method for calculating the theoretic or normal weight of infants.

Thus it would seem probable that up to a certain unknown optimum, the greater the amount of food properly balanced, the greater the growth. In the diet of the infant, undiluted milk mixtures furnish even more food than that used in the most modern dilutions of milk.

* Submitted for publication, Nov. 7, 1929.

* From the Department of Pediatrics, College of Medicine, State University of Iowa.

1. Wang, Chi Che; Hawks, Jean E., and Hayes, Bertha B.: Metabolism of Undernourished Children: Protein Metabolism, Am. J. Dis. Child. **35**:968 (June) 1928.

2. Boyd, J. D., and Nelson, Martha V.: Growth Studies of Children with Diabetes Mellitus, Am. J. Dis. Child. **35**:753 (May) 1928.

3. Daniels, Amy L.; Hutton, Mary K.; Stearns, Genevieve, and Hejinian, Lucea M.: Relation of Rate of Growth in Infants to Diet, Am. J. Dis. Child. **37**:1177 (June) 1929.

4. Finkelstein, H.: Lehrbuch Der Säuglingskrankheiten, Berlin, Julius Springer, 1921, p. 6.

Recent clinical reports [5] indicate that such feedings are of value for both the sick and the well infant. It has been noted that infants receiving undiluted milk increased in weight more rapidly than was expected when diluted milk was employed. Increases in weight alone would be of little significance unless something of the probable character of that increase were known. Whether this increase is merely the acquisition of adipose tissue due to the high energy value of the food ingested, or whether it represents better muscle and bone growth as well, can be demonstrated only by metabolic study.

It is my purpose in this paper to report observations on the growth and nitrogen retentions of nine well infants receiving undiluted mixtures of milk. The observations of the metabolism of calcium and phosphorus will be reported in another paper.

PROCEDURE

Nine male infants were kept under constant observation in a metabolism ward from the time they were 2 weeks of age until they were 6 to 10 months. Anthropometric measurements and roentgen studies of development of the bone were made each month. Metabolic observations were obtained at frequent intervals. A total of seventy-four such observations is herein reported.

Each of the infants received 660 cc. of milk at 2 weeks of age. This amount was increased gradually to 900 or 1,000 cc. at 6 months of age. Raw milk and corn syrup (6 per cent of the volume of the milk) were boiled vigorously for three minutes, cooled, and from 4 to 5 cc. of lactic acid [6] added to each quart of milk. Thirty cubic centimeters of orange juice was given daily until the infants were 3 months of age, and 60 cc. daily thereafter. One-half teaspoon of cod liver oil was given until 6 weeks of age, then 1 teaspoonful daily. Egg yolk was added to the feeding mixtures at age periods ranging from 3 to 6 months. Vegetables were added at 6 months and fruit sauces at 7 months. In order to preserve the uniformity of the diet, those receiving vegetables and fruit were given carrot and prune purées during each metabolic observation. No increases in the feeding mixture or additions to the milk diet were made less than from seven to ten days previous to the period of metabolic study. All feedings were prepared by the same person, and quantitative methods were employed. When the day's food was prepared, sufficient amounts were used to make one extra feeding for analysis. Any error in measurement or any variation in the materials used would thus be of no significance as the stated composition of the intake represents the chemical analysis of an aliquot of the food administered each day. After each feeding, from 10 to 20 cc. of distilled water was used to remove the curds adhering to the sides of the bottle, and these washings were fed to the infant.

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5. Marriott, McKim, and Schoenthal, Ludwig: An Experimental Study of the Use of Unsweetened Evaporated Milk for the Preparation of Infant Feeding Formulas, Arch. Pediat. **46**:135 (March) 1929. Brennemann, Joseph: The Curd and the Buffer in Infant Feeding, J. A. M. A. **92**:364 (Feb. 2) 1929.

6. No importance is attached to the presence of the acid other than its possible value in facilitating the digestion of milk.

TABLE 1.—*Nitrogen Data of the Infants Receiving Undiluted Milk*

Infant	Age Mo.	Age Days	Creatinine, Mg.	Weight, Kg.	Nitrogen Intake, Gm.	Nitrogen Output, Gm. Urine	Feces	Total	Nitrogen Retention Total Gm.	Per Kg. Gm.	Per Cent Retention
T. T.	1	22	52	4.86	2.91	1.78	0.15	1.93	0.98	0.201	32.6
R. W.	1	28	60	4.97	3.02	1.89	0.24	2.13	0.89	0.179	29.1
R. B.	2	0	..	5.21	4.35	2.91	0.46	3.60	0.98	0.188	22.5
K. H.	2	2	57	5.12	3.62	2.03	0.26	2.29	1.33	0.259	36.7
T. T.	2	5	54	5.29	3.65	2.05	0.30	2.35	1.30	0.245	35.3
R. W.	2	11	61	5.67	3.72	2.48	0.26	2.74	0.98	0.172	26.3
K. H.	2	15	59	5.60	3.72	2.61	0.27	2.88	0.84	0.150	22.5
M. R.	2	19	58	6.30	4.07	1.88	0.58	2.46	1.61	0.255	39.5
R. B.	2	19	54	5.77	4.35	2.44	0.38	2.82	1.52	0.264	35.0
R. W.	2	25	67	6.12	3.72	2.33	0.34	2.67	1.05	0.171	28.2
T. T.	2	26	60	6.08	3.98	3.03	0.29	3.32	0.66	0.108	16.5
R. S.	2	20	65	6.62	3.75	2.17	0.41	2.58	1.17	0.177	31.4
R. D.	3	0	60	5.65	3.75	2.15	0.39	2.54	1.21	0.214	32.2
R. B.	3	0	63	6.33	4.89	2.74	0.56	3.30	1.59	0.251	32.5
K. H.	3	0	65	6.08	3.84	2.80	0.40	3.20	0.64	0.105	24.5
C. T.	3	0	68	6.12	4.08	2.39	0.29	2.68	1.40	0.228	34.2
R. W.	3	7	72	6.66	4.20	2.68	0.49	3.17	1.03	0.155	24.5
T. T.	3	10	62	6.38	3.73	2.29	0.20	2.49	1.24	0.194	33.2
R. S.	3	10	70	6.80	4.57	2.90	0.61	3.51	1.06	0.156	21.0
K. H.	3	12	72	6.47	3.84	2.92	0.31	3.23	0.61	0.094	15.8
C. T.	3	14	72	6.40	4.33	2.67	0.29	2.93	1.37	0.214	31.6
R. B.	3	15	71	6.77	4.79	2.84	0.42	3.26	1.53	0.225	31.9
R. D.	3	20	61	6.10	4.04	2.34	0.40	2.74	1.30	0.213	32.1
M. R.	3	21	75	7.60	4.75	2.70	0.35	3.05	1.70	0.223	35.7
R. W.	3	21	80	7.10	3.92	2.57	0.31	2.88	1.04	0.146	26.5
T. T.	3	23	62	6.81	4.63	3.09	0.32	3.41	1.22	0.179	26.5
C. T.	3	28	74	6.67	4.33	2.54	0.28	2.82	1.51	0.226	34.9
M. R.	4	0	76	7.98	4.48	2.91	0.48	3.39	1.09	0.136	24.3
R. D.	4	0	73	6.32	4.02	2.60	0.28	2.88	1.14	0.180	28.3
T. T.	4	5	69	7.20	4.63	3.01	0.32	3.40	1.23	0.171	26.5
R. W.	4	5	84	7.65	3.92	2.73	0.41	3.14	0.78	0.102	19.9
K. H.	4	10	86	7.40	4.58	3.02	0.28	3.30	1.28	0.173	27.9
H. D.	4	16	74	7.50	4.10	2.41	0.52	2.93	1.17	0.155	28.5
R. S.	4	17	80	7.22	4.22	3.11	0.36	3.47	0.75	0.103	18.4
M. R.	4	18	83	8.51	4.45	3.19	0.38	3.57	0.88	0.103	19.7
R. W.	4	19	88	8.10	4.63	2.52	0.49	3.01	1.62	0.200	34.9
R. D.	4	20	83	6.82	4.04	2.15	0.19	2.35	1.69	0.247	41.8
T. T.	5	0	76	7.80	4.63	2.83	0.53	3.36	1.27	0.163	27.4
R. W.	5	2	96	8.30	4.63	3.11	0.32	3.44	1.19	0.143	25.7
R. D.	5	3	83	7.19	4.53	2.79	0.31	3.10	1.43	0.198	31.5
K. H.	5	8	95	8.30	4.60	3.30	0.36	3.66	0.94	0.113	20.4
T. T.	5	11	77	8.15	4.66	2.99	0.31	3.30	1.36	0.154	29.1
H. D.	5	14	76	7.75	4.93	3.19	0.35	3.54	1.39	0.179	28.1
R. D.	5	17	94	7.60	4.57	3.25	0.24	3.49	1.08	0.142	23.4
K. H.	5	21	96	8.67	4.86	3.45	0.45	3.90	0.96	0.110	19.7
R. W.	5	17	98	8.58	4.65	3.18	0.44	3.62	1.03	0.120	22.1
T. T.	5	25	86	8.15	4.66	2.99	0.31	3.30	1.36	0.154	29.1
C. T.	6	0	79	7.40	3.84	2.75	0.33	3.08	0.76	0.103	20.1
R. D.	6	0	97	8.08	4.79	2.79	0.37	3.16	1.63	0.203	34.0
K. H.	6	5	96	9.00	4.86	3.52	0.33	3.85	1.00	0.111	20.5
T. T.	6	7	91	8.68	5.42	3.98	0.40	4.38	1.04	0.119	19.1
C. T.	6	12	90	7.69	5.43	3.77	0.20	3.97	1.46	0.189	26.8
H. D.	6	20	90	8.63	5.67	4.00	0.42	4.42	1.25	0.145	22.0
C. T.	6	26	93	7.90	5.24	3.40	0.32	3.72	1.52	0.190	29.0
R. B.	7	10	95	9.58	4.94	3.17	0.36	3.53	1.41	0.147	28.5
M. R.	7	0	96	9.78	5.06	3.57	0.39	3.96	1.10	0.112	21.1
H. D.	7	14	92	8.99	5.47	3.59	0.54	4.13	1.34	0.149	24.4
C. T.	7	17	95	8.40	5.24	3.27	0.20	3.47	1.77	0.210	33.7
H. D.	7	28	94	9.30	5.83	3.98	0.52	4.50	1.33	0.143	22.8
R. B.	8	0	110	9.80	5.35	3.80	0.22	4.02	1.33	0.134	24.6
C. T.	8	6	97	8.50	5.28	3.50	0.55	4.05	1.23	0.144	23.3
H. D.	8	10	96	9.50	5.43	3.45	0.45	3.90	1.53	0.160	28.1
R. B.	8	19	111	9.97	5.86	4.00	0.33	4.33	1.53	0.153	26.2
C. T.	8	22	97	8.70	5.29	3.73	0.34	4.07	1.22	0.140	23.0
H. D.	8	25	113	9.60	5.32	3.52	0.41	3.93	1.38	0.143	25.9
R. B.	9	4	116	10.20	5.35	4.08	0.46	4.54	0.81	0.079	15.1
H. D.	9	9	113	9.60	5.32	3.15	0.33	3.48	1.84	0.190	34.5
R. B.	9	16	116	10.50	5.76	4.22	0.43	4.65	1.11	0.105	19.2
M. R.	9	22	112	10.90	5.86	4.14	0.48	4.62	1.23	0.112	21.0
H. D.	9	23	114	9.23	5.43	3.54	0.51	4.05	1.38	0.141	25.4
R. B.	10	0	116	10.50	5.76	4.05	0.40	4.45	1.31	0.124	22.7
M. R.	10	6	112	11.00	5.17	3.82	0.38	4.20	0.97	0.088	18.7
H. D.	10	6	117	10.00	5.40	3.42	0.45	3.87	1.53	0.153	28.3
M. R.	10	19	118	11.30	5.76	3.78	0.42	4.20	1.56	0.138	27.0

The babies were placed on a modified Hoobler[7] frame for three day collections of excreta. Carmine was used to determine the beginning and the end of the fecal output of each observation period. The urine output for each twenty-four hours was analyzed separately. Determinations of the nitrogen content of food, urine and feces were made according to a modification of the Kjeldahl method.[8]

None of the children suffered any noteworthy digestive disturbances while under observation. Infections of the upper respiratory tract ("colds") accompanied by slightly elevated temperatures were prevalent during January and February. No studies were made when the infant had an elevated temperature or frequent stools associated with "colds." One infant (C. T.) entered the ward with a high temperature and had a mastoidectomy at 1 month of age. He was not made the subject of metabolic study until 3 months of age, one month after he was entirely well. The infants were out of doors for a portion of each day when weather conditions permitted.

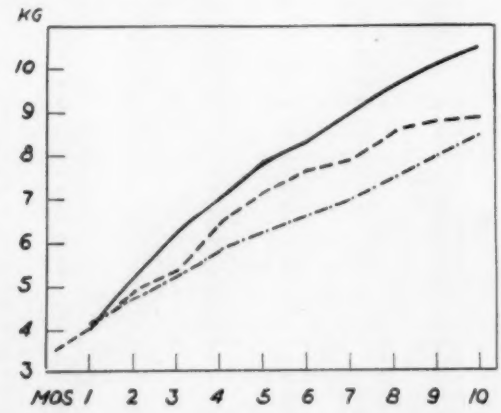

Chart 1.—Weight curve compared with accepted averages. The solid line represents the average weights at the different age periods of the infants fed undiluted milk; the broken line, the averages selected for comparison, and the dot and dash line, the "normal" weights as predicted from the birth weight according to Finkelstein.

RESULTS

Chart 1 shows the average weights of the infants compared with predicted weights calculated according to Finkelstein's formula[4] and with the averages for each age obtained by the Iowa Child Welfare Research Station.[9]

7. Hoobler, Raymond: An Adjustable Metabolism Bed for Infants and Young Children, Am. J. Dis. Child. **3**:253 (April) 1912.

8. Hawk and Bergein: Practical Physiological Chemistry, Philadelphia, P. Blakiston's Son & Company, 1926, p. 711.

9. Unpublished observations of the Iowa Child Welfare Research Station. These data have been selected for comparison throughout this study because the averages for both weight (chart 1) and length (chart 2) are greater than those previously published.

At 1 month of age the average weight of each of the groups was practically the same. After 2 months of age, the average weight of infants receiving undiluted milk mixtures exceeded the other values. At 4 months of age, the birth weight of this group had been doubled. After that time, the average weights of the infants used in study were from 8 to 18 per cent greater than the average weights accepted for comparison. There was practically no diminution in the rate of growth as the infants approached 10 months of age.

The growth in length of the infants is shown graphically in chart 2. At birth, the length of none of the infants exceeded 50.4 cm. At 1 month of age, the average length of the infants of this study was less than that of those of the Child Welfare Research Station group. After

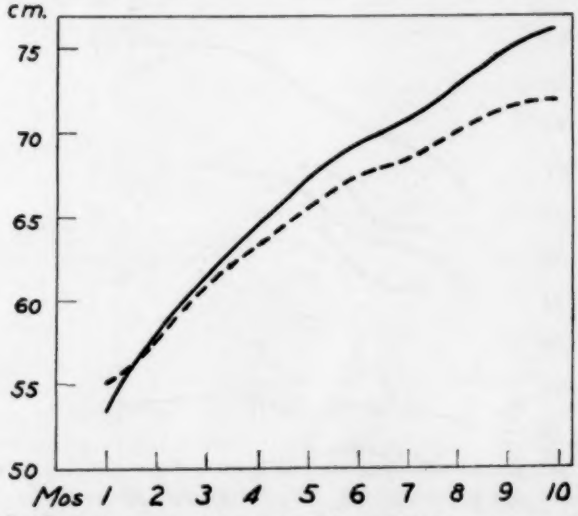

Chart 2.—Length curve compared with accepted averages. The solid line represents the growth in length of the infants fed undiluted milk; the broken line, the averages chosen for comparison.

2 months of age, the infants receiving undiluted milk surpassed the accepted averages and the increased increment of growth became more marked as the infants grew older. The increase in length (15.5 cm.) during the first six months was practically equivalent to the increase (15.9 cm.) during the first ten months in the average lengths used for comparison.

NITROGEN RETENTION

Some information concerning the nature of this increased growth can be obtained by a study of the nitrogen retention of the infants.

In chart 3, the average total nitrogen intake, output and retention of the children of this study are compared with recently published

averages [3] obtained from infants who received modern dilutions of milk. The nitrogen intake of the infants who were fed undiluted milk exceeded that of the group receiving diluted milk. While the nitrogen output of the first group was greater, the difference between the output of the two groups was much less than the difference between the intake. Thus the total amount of nitrogen retained per day was greater when an increased quantity of milk was fed. Not only was the total amount of nitrogen retained increased, but the amount retained represented a greater percentage of the intake than did that of the infants receiving less food. The average retention of the infants receiving diluted milk was 19.5 per cent of the intake, while that of the infants receiving undiluted milk was 26.9 per cent.

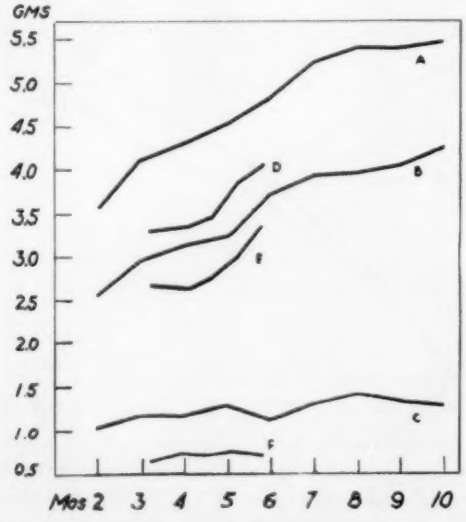

Chart 3.—Curves *A*, *B* and *C*, respectively, represent the average total daily nitrogen intake, output and retention of the infants fed undiluted milk. Similarly *D*, *E* and *F* represent the intake, output and retention of infants receiving diluted milk. The averages for infants fed diluted milk were obtained by grouping those metabolic periods showing a similar creatinine output. The averages for the infants fed undiluted milk were obtained by grouping according to age. The period from 1 month, 16 days to 2 months, 15 days is recorded as 2 months. If the data for undiluted milk were rearranged and grouped according to creatinine output the difference between the averages for the two studies would not be decreased.

It is conceivable that when there was such an increase in the weight of the infants as demonstrated in chart 1, the nitrogen retention per unit of body weight might have been decreased even though the total nitrogen retention was greater than that of infants fed less food. This did not result, however, as the total quantity of nitrogen retained was

such that the retention per kilogram at every age was greater than that reported for infants fed diluted milk.

The total quantity of nitrogen retained per day did not differ markedly at the different age levels. Hence the amount retained per unit of body weight was a gradually decreasing quantity as the infant grew older (from 199 mg. of nitrogen per kilogram at 2 months of age to 123 mg. at 10 months of age; see chart 4).

COMMENT

For many years the food requirements of infants were calculated on the basis of their so-called theoretic weight, for the calculation of which Finkelstein's formula [4] has been used. The milk intake frequently has been approximately 10 per cent of this theoretic weight. Had the infants of this study been fed on such a basis, the food intake would

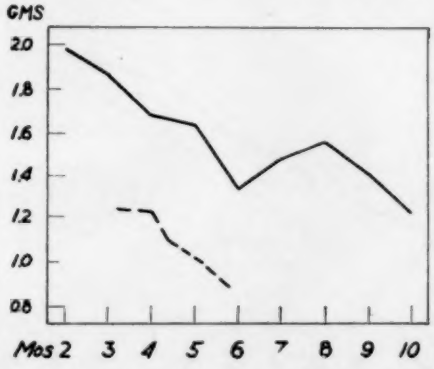

Chart 4.—The solid line shows the average nitrogen retention per kilogram at the different age periods of the infants fed undiluted milk. The broken line represents the average nitrogen retention of infants fed diluted milk.

have been decreased by about one-third (chart 5). It is probable that an infant so fed cannot be expected to attain the optimum growth. As the standards for growth and development become obsolete, so also do the ideas of food requirements. Many requirements are based on the minimum requirements for life rather than for optimum development. The relation of the optimum to the minimum and maximum growth is yet to be determined.

The intake of the infants of my study was not calculated on the basis of any assumed weight gain. At 2 weeks of age the children received feedings of undiluted milk, six bottles each holding 4 ounces. The type of food and the proportions between the constituents remained unchanged.[10] The quantity was increased until four feedings of 8 ounces

10. The amount of nitrogen retained by an infant on a constant diet varied greatly. A change of type of food was considered inadvisable as these variations might seem to follow the changes of diet and yet be wholly unrelated.

each (or the equivalent) were being given at 7 months of age, and the infant was allowed to gain as he would.

It is apparent from charts 1 and 2 that the nine well infants who were so fed grew more rapidly in weight and length than was predicted by the highest available averages. Any conclusions drawn from such a small number of cases must be modified by a consideration of the factor of chance due to the method of sampling employed. Table 2 shows the difference, probable error and their quotient for both the weight and the length of the infants of this study compared with the averages of the Child Welfare Research Station. Pearl[11] stated that after the number of cases has been taken into consideration a quotient of 3 or more represents a signicant difference. At 1 month of age the infants of this study were below the averages used for comparison in both weight and length. Both quotients increased with the age of the child,

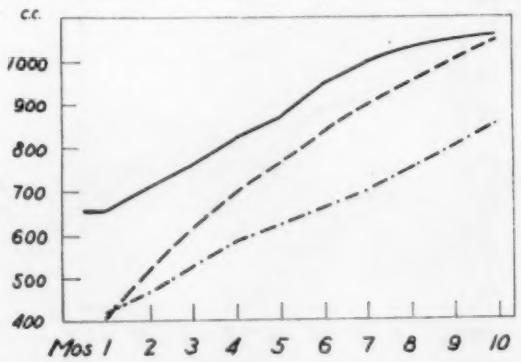

Chart 5.—Quantity of milk ingested compared with accepted standard. The solid line represents the milk intake at the different ages of the infants fed undiluted milk; the dot and dash line, the quantity they would have received had they been fed 10 per cent of their theoretic weight calculated according to Finkelstein. The broken line is purely hypothetic, representing the quantity of milk equivalent to 10 per cent of the actual weight of the infants at the different age periods.

as is shown graphically in chart 6. After due discount has been made for the limited number of cases and the variations, the difference is still of too great magnitude to be discounted as a factor of chance. This would indicate that the increase in food intake enabled the infants to attain a growth in excess of that predicted from accepted averages.

Since milk forms the basis of the infant's diet, increases in the total nitrogen intake accompany the increase in the total food intake. The

11. Pearl, Raymond: Medical Biometry and Statistics, Philadelphia, W. B. Saunders Company, 1927, chapter 10.

nitrogen requirement of the infant never has been satisfactorily determined. The early work of Hoobler [12] was interpreted to indicate that great dangers may result from excessive protein feeding. Talbot and

TABLE 2.—*Difference Between the Average Weights and Lengths of Infants Fed Undiluted Milk and the Averages Compiled by the Iowa Child Welfare Research Station, the Probable Error of this Difference and Their Quotient* Calculated According to Pearl (Medical Biometry and Statistics)*

Age, Months	Weight			Length		
	Difference, Kg.	Probable Error	Quotient	Difference, Cm.	Probable Error	Quotient
1	0.08	0.12	0.7	—1.58	0.43	—3.6
2	0.30	0.11	2.5	+0.13	0.37	+0.35
3	0.80	0.15	5.1	0.21	0.66	0.32
4	0.54	0.21	2.6	0.85	0.84	1.0
5	0.64	0.14	4.5	1.44	0.64	2.2
6	0.69	0.12	5.1	1.74	0.52	3.3
7	1.14	0.16	7.1	3.22	0.50	6.1
8	1.00	0.16	6.3	2.55	0.71	3.5
9	1.36	0.22	6.1	3.54	1.06	3.3
10	1.66	0.20	8.3	5.25	0.85	6.1

* Formula for determining quotient $\frac{A - B}{\sqrt{(\text{Probable error of A})^2 + (\text{Probable error of B})^2}}$

A = The average weight or length of the infants of this study.
B = The averages of the Iowa Child Welfare Research Station.

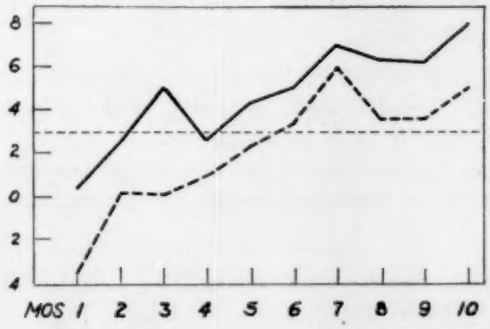

Chart 6.—The plotted weight (solid line) and length (broken line) quotients of table 2, showing graphically by the increasing value of the quotient that the increment of growth of the infants fed undiluted milk was greater than that of the average selected for comparison.

Gamble [13] were unable to reproduce Hoobler's results by high protein feeding. Raw milk was used in these studies and the difficulties encountered were those of digestion.

12. Hoobler, Raymond: The Protein Need of Infants, Am. J. Dis. Child. **10**:153 (Sept.) 1915.

13. Talbot, Fritz B., and Gamble, James L.: Protein Metabolism of an Infant, Am. J. Dis. Child. **12**:333 (Oct.) 1916.

Keller [14] reported comparative nitrogen retentions of several infants receiving dilutions of breast milk and of cow's milk. These infants were markedly malnourished and the food intake so much less than any in general use at the time this paper was prepared that the data cannot be compared with those of recent studies.

The figures on nitrogen intake reported by Daniels [8] in the study aforementioned are greater than those previously published. The nitrogen intake is increased still further in these infants by the use of feedings of undiluted milk. No digestive upsets or undesirable effects have been noted. This study would confirm previous clinical observations that feedings of undiluted milk are well tolerated and would indicate that the increase in weight is a desirable one and not merely the acquisition of adipose tissue.

SUMMARY

1. Studies on the growth and nitrogen retention of nine well infants receiving feedings of undiluted milk are reported.

2. Additional evidence is presented that growth is definitely influenced by the food intake. The increase both in weight and in length of the infants was too great to be discounted as a factor of chance.

3. The total nitrogen retention, retention per kilogram and the percentage of the intake retained were greater than those of infants studied under similar conditions but receiving less food.

4. These results are interpreted to indicate that feedings of undiluted milk not only are well tolerated, but that such feedings produce an increased rate of growth both in weight and in length. This increase is accompanied by a proportionate increase in the retention of nitrogen and therefore does not represent merely the accumulation of adipose tissue.

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14. Keller, Arthur: Phosphor und Stickstoff im Sauglingsorganismus, Arch. f. Kinderh. **29**:1, 1900.

UROBILINURIA

ITS PROGNOSTIC VALUE IN CHILDREN WITH HEART DISEASE *

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Since the discovery of urobilin, in 1868, by Jaffe, urobilinuria has been observed in a wide variety of clinical conditions. Although numerous hypotheses have been proposed to explain the origin of urobilin in both the normal and the diseased organism, information of positive value was unavailable until within the last few years. Peyton Rous and his collaborators have approached anew the entire problem of the physiology and pathology of urobilin. Their work includes the elaboration of a quantitative method for determining urobilinuria, described by Elman and McMaster.[1] This method possesses the two essential requisites for a clinical laboratory procedure, simplicity and accuracy, and it was adopted for the studies reported herein.

In a series of articles, Elman and McMaster presented abundant evidence to support the view that in the uninfected animal the intestinal tract is the only site of origin of urobilin. Urobilinuria is an expression of the inability of the liver cells to remove from the circulation the urobilin brought to the liver by the portal blood. The pigment passes on into the systemic circulation to the kidneys and is excreted in the urine. Urobilinuria was never found after damage to the liver except when the bile pigment was present in the intestines.

From these experiments, it can be concluded that theoretically a urobilinuria may result from two causes: (1) an increased production of bilirubin as in hemolysis and (2) a derangement of the function of the liver cells. The second cause operates to produce the urobilinuria observed in patients with cardiac lesions. The urobilinuria in congestive cardiac failure may be dependent on two conditions: a slackening of the

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* From the Department of Pediatrics and the Department of the Laboratories, New York Post-Graduate Medical School and Hospital.

1. Elman, R., and McMaster, P. D.: Studies on Urobilin Physiology and Pathology: I. The Quantitative Determination of Urobilin, J. Exper. Med. **41**: 503, 1925; IV. Urobilin and the Damaged Liver, ibid. **42**:99, 1925. McMaster, P. D., and Elman, R.: II. Derivation of Urobilin, J. Exper. Med. **41**:513, 1925; III. Absorption of Pigments of Biliary Derivation from the Intestines, ibid, **41**: 719, 1925; VI. The Relation of Biliary Infections to the Genesis and Excretion of Urobilin, ibid., **43**:753, 1926.

rate of flow of blood through the liver (a circulatory stasis) and a diminished oxygen supply to the liver cells. The latter cause is suggested by the observations on the unsaturation of oxygen of the venous blood in congestive heart failure by Lundsgaard,[2] Schmitz and Killian.[3]

The cases studied in this investigation included those of patients who were confined to the babies' wards and children who were attending the cardiac clinic of the pediatric department. Varying degrees of cardiac impairment were represented among these patients, from grave decompensation to lesions with no apparent clinical symptoms in children whose activities were unrestricted. A total of 5,000 determinations of the urobilinuria were made in 700 cases. To simplify the presentation of the results, these cases were divided into four groups: 1. The control group of 350 apparently healthy children who were admitted to the hospital wards for the removal of tonsils and adenoids. 2. The comparative group of 32 noncardiac children confined to bed and representing various pathologic states. 3. The ambulatory group, made up of 152 patients attending the cardiac clinic. 4. The decompensated group (cases with congestive cardiac failure) composed of 50 cases presenting symptoms of varying grades of cardiac decompensation.

Determinations of the urobilin were made on specimens of urine collected during twenty-four hours, and the results were expressed in terms of concentration, i. e., number of milligrams per hundred cubic centimeters, and as the total output for this period.

Chart 1 summarizes the results for the occurrence of urobilinuria in the four groups of patients. In 15 of 350 control cases, or 4 per cent, there were positive reactions for urobilinuria. With the exception of 2 instances, the concentration of urobilin varied from 3 to 9 mg. per hundred cubic centimeters. In this group, estimations of urobilin were made on casual, preoperative specimens of urine voided between 9 a. m. and 12 m. Bauman[4] stated that urobilin is present in the normal urine in negligible quantities. Many investigators are agreed on the fact that urobilin is present in the urine of the normal subject; however, considerable variation is evident in the quantities found. During a twenty-four-hour period, Gerhardt found from 7.4 to 7.9 mg.; Friederick Müller, as high as 20 mg.; Saillet found from 30 to 130 mg. and Hoppe-Seyler, from 80 to 140 mg.[5] These deviations

2. Lundsgaard, C.: Studies of Oxygen in the Venous Blood: II. Studies of Oxygen Unsaturation in Venous Blood of a Group of Patients with Circulatory Disturbances, J. Exper. Med. **27**:179, 1918.

3. Schmitz, H. W., and Killian, J. A.: Oxygen Unsaturation of Venous Blood in Cardiac Failure, Arch. Path. **5**:925 (May) 1928.

4. Bauman, L.: Urobilin and Urobilinogen. Endocrinology and Metabolism, New York, D. Appleton & Company, 1922, vol. 3, p. 165.

5. Quoted by Marcussen and Hansen (footnote 6).

observed for the normal output of urobilin are probably due to sources of error in the analytic methods, of which some have been pointed out by Marcussen and Hansen.[6]

Two of the control subjects gave unusually high figures for the excretion of urobilin. In one patient, the concentration of urobilin was 94 mg. per hundred cubic centimeters, and simultaneously an icterus index of ten was noted. Since at this time tonsillectomy was deemed unwise; the child was referred to the outpatient department for detailed study. After observation there for two weeks, the patient

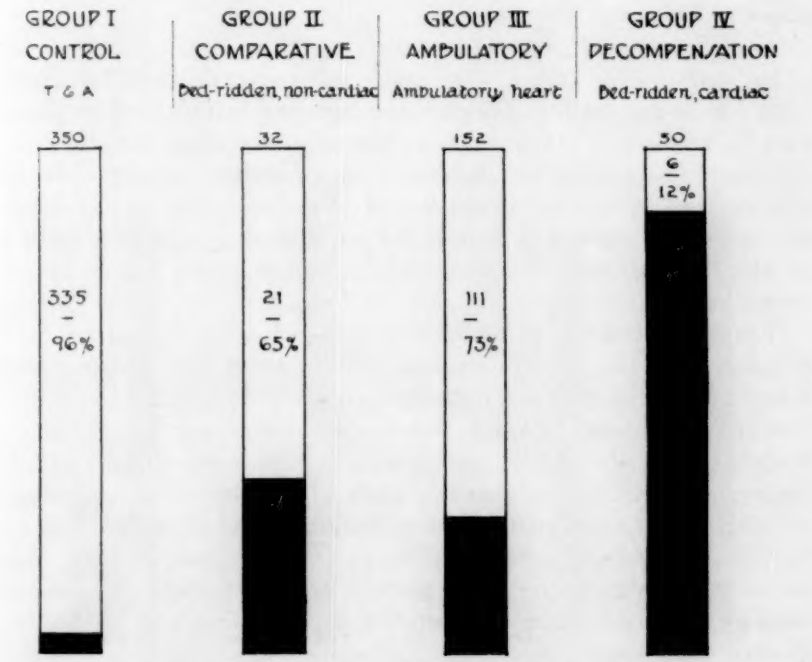

Chart 1.—A group study of 584 cases summarizing the results in the occurrence of urobilinuria in the four groups of patients.

was readmitted at a time when the icterus index had returned to normal and no urobilin was found in the urine. Tonsillectomy was performed without a resultant urobilinuria. In the other case, 188 mg. of urobilin per hundred cubic centimeters of urine was found; however, the tonsils were removed before any further studies were made, and the tonsillectomy was not accompanied by any untoward results nor by an increased urobilinuria. An intensive clinical study of these two children offered no explanation of the urobilinuria.

6. Marcussen, S., and Hansen, S.: On the Determination of Urobilin in Urine, J. Biol. Chem. **36**:381, 1918.

The influence of tonsillectomy and adenectomy on the urobilinuria was studied in fifty of the control cases by determining the urobilin of the urine voided before and after operation. Forty-nine patients gave negative reactions at both times. One, however, showed a urobilinuria of 9 mg. per hundred cubic centimeters for the twenty-four hours after tonsillectory, although before operation no urobilinuria was noted. Forty-eight hours after operation the urobilinuria had disappeared and remained negative for the following week. In this instance the urobilinuria may have been due to postoperative bleeding, which diminished the hemoglobin to 55 per cent (by Sahli), or to a suspected endocarditis.

Urobilinuria was observed in eleven, or 35 per cent of the patients in the comparative group. The concentration of the urobilin varied from 3 to 24 mg. per hundred cubic centimeters. Included in this group were 32 noncardiac but bedridden children of the usual hospital type, of whom many were in pathologic states usually accompanied by urobilinuria. A detailed discussion of the urobilinuria in this group will be omitted since it is outside the scope of this paper, the subject of which is the study of urobilinuria in children with lesions of the heart.

Under the heading of ambulatory cases of heart disease we have classified the cases of 150 children who attended the cardiac clinic. It had been planned to determine the excretion of urobilin in twenty-four hour specimens of urine, but in this attempt we were seriously handicapped by the lack of intelligent cooperation on the part of the mothers. Since there was always a doubt whether the volume submitted for analysis was a twenty-four hour collection, the results of the analyses have been expressed in terms of concentration, rather than of total output. In this group forty-one patients, or 27 per cent, gave positive reactions for urobilin, the concentration varying from 3 to 24 mg. per hundred cubic centimeters.

The decompensated group included 50 children who were confined to bed with varying grades of congestive cardiac failure. In this class forty-four, or 88 per cent, had a urobilinuria, ranging from 3 to 586 mg. per hundred cubic centimeters, or from 6 to 865 mg. per day. The results of a comparative study of urobilinuria in these four classes are presented graphically in chart 2. It is evident from chart *2I* that the highest percentage of occurrence of urobilinuria was observed in the decompensated group, but that urobilinuria was less frequent among the ambulatory cases of heart disease (compensated) than among the miscellaneous patients in the wards. Moreover, in chart *2II* it is seen that also in this decompensated class the maximum output of urobilin was found. In the construction of this chart, the urobilinuria has been uniformly reported in terms of concentration in order to make

all of the results comparable. The maximum urobilinuria in the decompensated group is about twenty-five times greater than the maximum for the ambulatory and the miscellaneous classes. Operative procedures (fig. *III*, chart 2) were followed by an increased urobilinuria in 86 per cent of the children with decompensated cardiac disease, but in only 2 per cent of the control cases. Moreover, the intensity of the urobilin

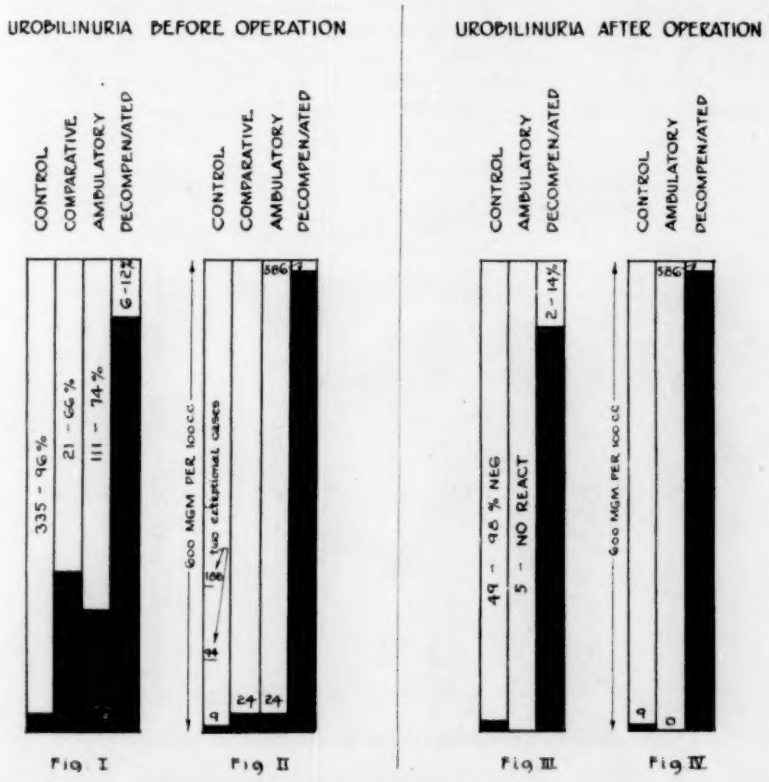

Chart 2.—A comparative study of the 584 cases; the urobilinuria in patients before operation and the urobilinuria in patients after operation.

reaction postoperatively was greatest in the decompensated group (fig. *IV*, chart 2.)

In an attempt to estimate the prognostic significance of the urobilinuria in congestive cardiac failure, variations in the excretion of urobilin were compared with the clinical course of the disease. Results for some of these observations were presented graphically in the subsequent charts. Curves for the urobilinuria were drawn on the basis of the total output for a twenty-four hour period.

REPORT OF CASES

CASE 1.—The graphs in chart 3 present the fluctuations in the urobilinuria of M.S., aged 12, who was admitted to the hospital because of double mitral lesions, auricular fibrillation with severe grade of decompensation and generalized edema. On admission, the patient excreted 118 mg. of urobilin during twenty-four hours, and digitalis therapy was immediately instituted and was continued for three days. During this period of rest in bed and digitalis therapy, there was observed a continuous fall in the output of urobilin. The decrease in the urobilinuria continued for four days after treatment with digitalis was instituted and during rest in bed, ultimately only a trace of urobilin being noted. A general clinical improvement accompanied the fall in urobilinuria. Subsequently the symptoms of congestive failure reappeared and paralleled the impairment of cardiac function, after which the excretion of urobilin rose to a level and reached a maximum of 865 mg. per

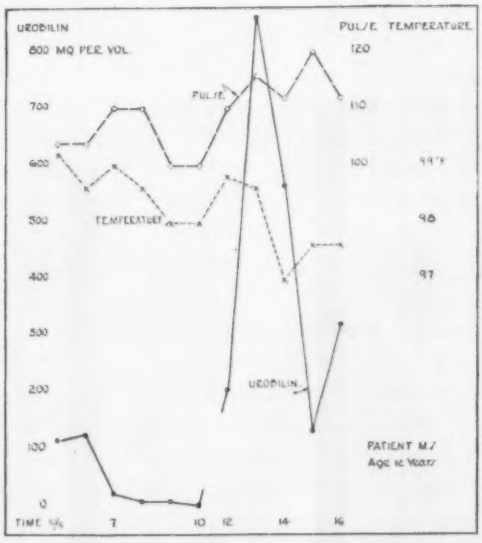

Chart 3.—Fluctuation in the urobilinuria in case 1

twenty-four hours. A second period of digitalis therapy produced a fall in the urobilinuria within two days to 130 mg. per twenty-four hours, but this fall was followed by a rise to 320 mg. during the day of death. The curve for the pulse rate shows a fairly steady rise for the last five days of life, but this rise is much less striking than is the precipitate increase in the excretion of urobilin.

CASE 2.—L. C., aged 9, was admitted to the hospital with a chronic cardiovalvular disease, a double mitral lesion with marked hypertrophy, moderate cyanosis and dyspnea on exertion. The history of cardiac diseases dated back to three years before admission, when he had an attack of rheumatic fever. Six weeks before admission the child had scarlet fever, and four weeks before admission he had pneumonia with effusions. It was observed that while digitalized the patient's clinical condition was fair, but when the administration was discontinued, he became acutely decompensated. Hence, throughout his entire stay in the hospital, the child was maintained on treatment with uniform amounts of digitalis. During

the first seven days after admission, the patient was confined to bed, and for this period there were considerable fluctuations in the urobilinuria and also in the pulse rate, and the excretion of urobilin varied from 0 to 115 mg. for twenty-four hours (chart 4). From the eighth day until death, the child was occasionally permitted to leave his bed, and to rest in a chair, or to walk about for intervals of from one to two hours, and on one occasion for an entire day, but these periods of mild exercise were apparently without any demonstrable effect on the urobilinuria. A transfusion of 500 cc. of blood on the twentieth day was followed by a prompt rise in the urobilinuria to 115 mg. per hundred cubic centimeters. During the subsequent twenty-four hours, the amount of excretion of urobilin fell to a mere trace, but it again rose to 241 mg. the third day after transfusion. A similar increase in urobilinuria was noted after a second transfusion and after the output of urobilin reached 112 mg. per hundred cubic centimeters. The levels reached by the

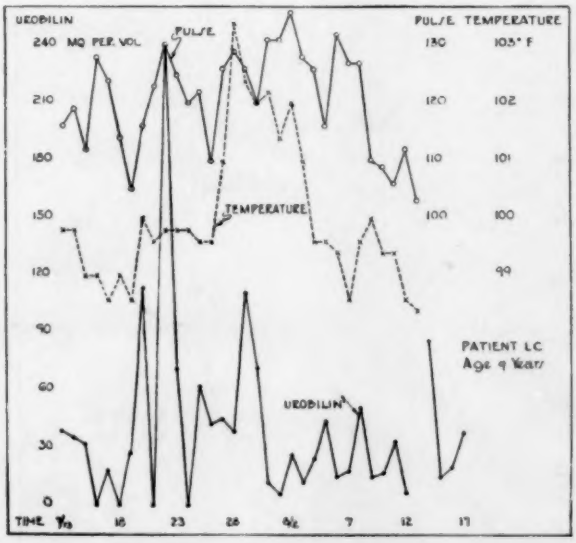

Chart 4.—Fluctuation in the urobilinuria in case 2.

urobilinuria immediately after transfusions are conspicuously outstanding peaks on the urobilin curve, and it appears probable that there may have been a dependence as between cause and effect, of this increased urobilinuria on the transfusions. A furuncle appeared on the neck of the patient, and six days later an incision was made on it and the pus drained. Simultaneously symptoms of grave congestive failure and edema developed. An increase of 6 pounds in (2.7 Kg.) body weight was noted. The urobolinuria rose to 85 mg. per hundred cubic centimeters, but fell within twenty-four hours to 15 mg. with a final increase to 38 mg. before death.

The temperature curve fluctuated between 98.5 and 100 F., except for a marked rise to 103.4 F. at the time of the second transfusion. The curve for the pulse rate was fairly synchronous with the urobilinuria curve.

Case 3.—E. L. (chart 5), 5 years and 4 months of age, was admitted to the hospital for chronic cardiovalvular disease, double mitral lesion and aortic insuf-

ficiency, marked hypertrophy, anemia, edema of the extremities, cyanosis, dyspnea and orthopnea. On admission, when the patient had been confined to bed, a fall in urobilinuria was noted, but on the ninth and fourteenth days after admission a myringotomy was performed, and on those days the excretion of urobilin mounted to 46 mg. per hundred cubic centimeters. Again on the day of the tonsillectomy, the sixteenth day after admission, and on the following day there was observed a precipitate rise in the urobilinuria to 80 mg. per hundred cubic centimeters. Digitalis therapy was begun and continued until the time of death. On the nineteenth day, a transfusion of 175 cc. of blood was made. An increase in the urobilinuria to 94 mg. per hundred cubic centimeters promptly appeared. The following day 244 mg. of urobilin was excreted, and on the third day following the transfusion, the day of death, the urobilinuria had reached 677 mg.

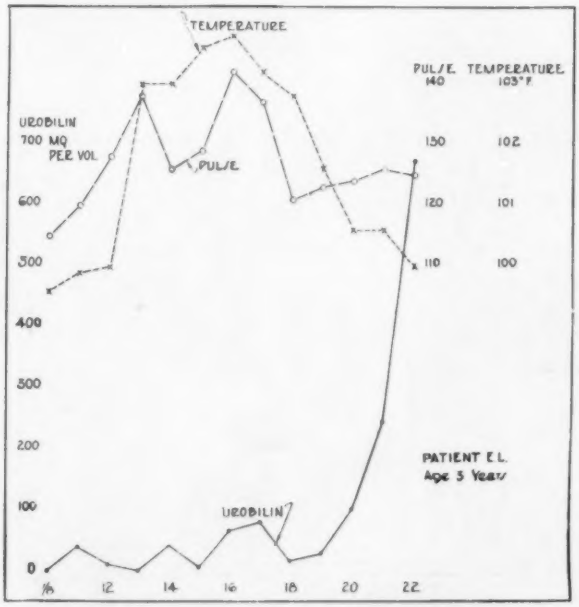

Chart 5.—Fluctuation in the urobilinuria in case 3.

This patient showed a definite urobilinuria after myringotomies and tonsillectomy. During this period, the temperature persisted at a high level. A pronounced urobilinuria followed the transfusion, but for the final six days of life the temperature continued to fall. The pulse rate curve shows peaks that correspond to the increased urobilinuria after myringotomy and tonsillectomy, but there was but little change in the pulse rate for the last four days of life.

CASE 4.—L. F. (chart 6), aged 5 years and 6 months, was admitted to the hospital for a double mitral lesion resulting from rheumatic fever six months previous to admission, marked hypertrophy with chronic passive congestion of the lungs, spleen and liver and dyspnea. Throughout his entire stay in the hospital, the patient was confined to bed. For three days after admission, the output of urobilin varied from a trace to 14 mg., but coincident with an asthmatic attack the urobilinuria rose to 32 and then to 40 mg. per hundred cubic centimeters for

twenty-four hours. A fall in the urobilinuria to the original level followed the subsidence of the asthmatic symptoms. On July 21 symptoms of grave decompensation, dyspnea and peripheral edema developed and persisted for twelve days. A marked increase in the excretion of urobilin was noted during this period, and it reached its maximum at 520 mg. on July 30. Digitalis therapy was instituted, and for forty-eight hours, on July 27 and 28, there was an apparent improvement in the patient's clinical condition, and, paralleling this improvement, the urobilin fell to 131 and then to 9 mg. per hundred cubic centimeters per day. After August 1, the amount of urobilin diminished, reaching a mere trace on August 6 and 7. Coincident with this decline in urobilinuria, a lessening of the peripheral edema and of the dyspnea was noted. However, about August 8 symptoms of acute and

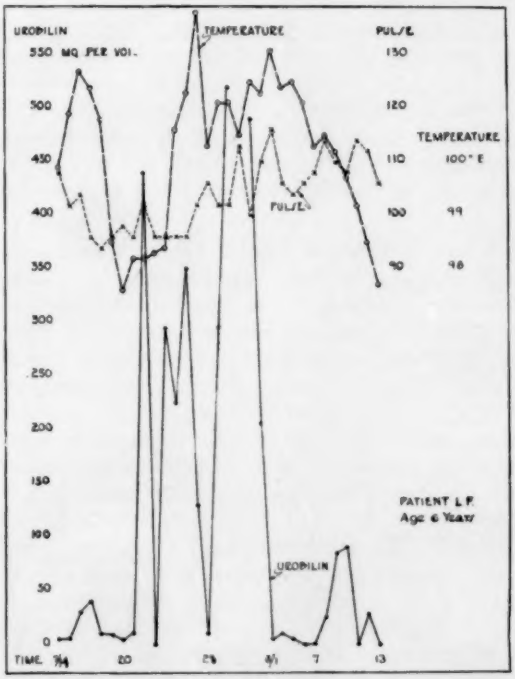

Chart 6.—Fluctuation in the urobilinuria in case 4.

grave congestive failure reappeared and continued until August 12. The patient became dyspneic, cyanotic, edematous and stuporous. The excretion of urobilin rose from a mere trace to 26 mg., then to 85 mg., and reached its maximum at 89 mg. per hundred cubic centimeters, subsequently dropping on August 13 to a mere trace again. From August 13 to September 23, on which day the child left the hospital, his clinical condition remained fairly constant, characterized by little edema and an absence of dyspnea when he was in the upright position. Throughout this period the urobilinuria persisted at a low level, from mere traces to 12 mg. per hundred cubic centimeters, but on one occasion, on September 14, it rose to 47 mg. The fluctuations in excretion of urobilin in this instance were enormous from day to day, but these variations reflected well the patient's clinical condition. In this chart, there is a significant lack of parallelism between the curves for

urobilinuria and for the pulse rate. There is also a lack of analogy between the curves for urobilinuria and for the body temperature.

CASE 5.—E. B. (chart 7), aged 8 years, admitted to the hospital on July 6 for chronic cardiovalvular disease and double mitral lesion with marked hypertrophy but good compensation. From July 21 to August 4 the amount of urobilin persisted at a low level, from a mere trace to 9 mg. per hundred cubic centimeters, but the incidence of otitis media on August 2 was accompanied by a hyperurobilinuria to 52 mg., which continued for forty-eight hours. Throughout this period, urobilin was absent from the urine or occurred in traces only, and on the days it occurred, the patient was wholly confined to bed. However, on the days on which the patient spent part of the time out of bed, definite increases in urobilin were demonstrable.

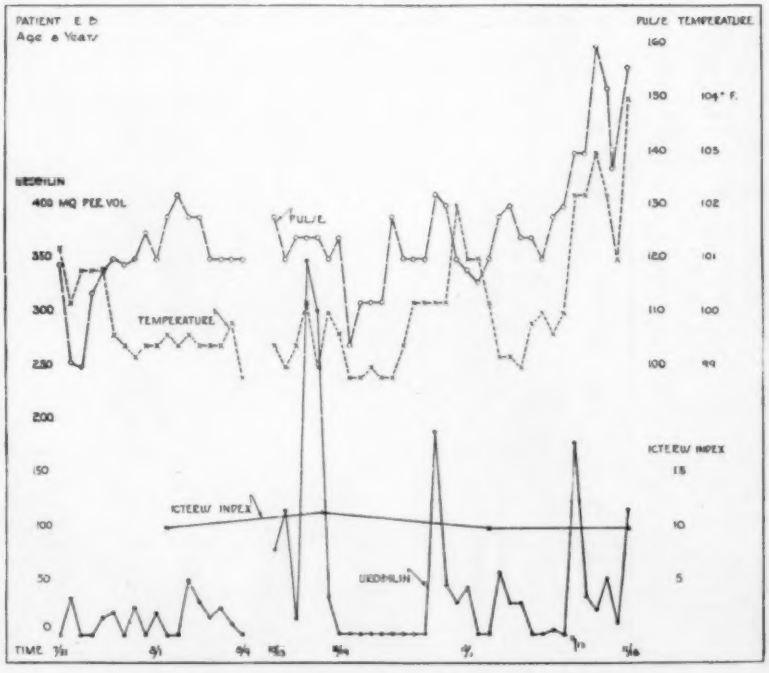

Chart 7.—Fluctuation in the urobilinuria in case 5.

On August 10, the patient was sent to the country and remained there until readmitted to the hospital on October 12. At the time of the second admission, symptoms of congestive failure were manifest. There was evident general edema; the body weight was 56 pounds (25.4 Kg.), whereas during her previous stay in the hospital, the body weight had not exceeded 50 pounds (22.7 Kg.) For the seven days of impaired cardiac function, indicated by the existence of the edema and dyspnea, a hyperurobilinuria persisted, and reached its maximum at 350 mg. per hundred cubic centimeters. At the end of seven days, a fair degree of compensation was effected with digitalis therapy, and the amount of urobilin fell to a mere trace paralleling this improvement. The body weight was diminished to 48 pounds (21.8 Kg.). During the following five days, the patient was kept in bed

for the entire day. On the sixth day the child was allowed out of bed for one hour, on the seventh day for two hours, and the entire eighth day was spent out of bed. Hyperurobilinuria appeared on the ninth day and continued for four days. This increase in the amount of urobilin was interpreted as signifying the necessity of restricting the patient's activity, consequently the child was again confined to bed for the following three days. During this interval, a fall in urobilinuria was noted, but when the patient was again permitted to leave her bed on November 3, the urobilinuria rose to 59 mg. per hundred cubic centimeters and remained moderately increased for three days. Restriction of activities of the patient in bed produced a drop in the amount of unrobilin that was excreted to within physiologic limits. On November 11, 12 and 13, the child spent about five hours each day on the roof of the nursery. Here her activities were unlimited. During the day of November 13, 188 mg. of urobilin were excreted. The patient was returned to bed in the ward and pericarditis was clinically demonstrated. For the subsequent three days, the urobilinuria diminished, but there was a terminal rise to 118 mg. per hundred cubic centimeters on the day of death.

In these observations, although there are variations of pulse rate and temperature in the same direction as the urobilinuria, the curves for pulse rate and temperature do not parallel the urobilin curve in time or magnitude. We believe that this case illustrates the value of the urobilinuria in regulating the activities of a child with cardiac disease.

Case 6.—A. P. (chart 8), aged 13, was admitted December 21, for chronic cardiovalvular disease, mitral stenosis and insufficiency, pericarditis with effusion, extreme orthopnea and dyspnea. The history of cardiac disease extended back over a period of two years. The patient on admission was confined to bed and digitalis therapy was instituted. During the first five days the excretion of urobilin fell from 1,782 to 32 mg. per hundred cubic centimeters for twenty-four hours. A corresponding drop in the pulse rate was also noted, but the temperature curve was behind both the pulse rate and urobilin curves. Orthopnea and dyspnea diminished, and the patient appeared more comfortable. A hyperurobilinuria appeared on December 28, and persisted with marked daily variations until January 5. The variations in the excretion of urobilin were from 40 to 1,120 mg. per twenty-four hours. A rise in the pulse rate accompanied the hyperurobilinuria. However, an initial fall followed by a rise, was observed in the temperature. During these seven days the child was delirious and refused to eat. On January 9, urobilinuria was diminished to mere traces, and continued at this low level until January 29. The fall in urobilinuria was followed by disappearance of clinical signs of decompensation. In order to condense the chart, graphs for the twenty-one days were omitted. The temperature varied from 99 to 100 F., and the average pulse rate was 90. The patient spent the greater part of the day out of bed and was engaged in unrestricted activities about the wards and nursery.

For the forty-eight hours of January 30 and 31 a rising urobilinuria appeared, which reached 660 mg. per hundred cubic centimeters on February 1. On this day the patient became restless and dyspneic and complained of severe pain in the region of the heart. A simultaneous rise in the pulse rate and temperature was noted during these four days. From February 1 to February 14, the child was confined to bed. Hyperurobilinuria persisted during the period, showing marked variations from 50 to 720 mg. per twenty-four hours. It is interesting to note that, despite the increasing urobilinuria from February 6 to 9, both the temperature and pulse rate continued to decline to their lowest levels. Subsequently, both of these curves rose during a period of diminished urobilinuria. Clinical symptoms of congestive failure subsided on February 14 and did not reappear before April

29, when the patient was discharged as improved. Urobilinuria continued but at a relatively low level from February 14 to 24, ranging from slight traces to 65 mg. for twenty-four hours. During the remainder of the patient's stay in the hospital, the urobilinuria never exceeded physiologic limits.

CASE 7.—H. M. (chart 9), aged 12 years and 6 months, was admitted to the hospital for chronic cardiovalvular disease with acute endocarditis, marked hypertrophy, mitral stenosis, regurgitation and pericarditis with effusions. Digitalis therapy was initiated at the time of admission and was continued until July 30. The child was up and around the wards for the greater part of the day on July 7, and during this day 371 mg. per hundred cubic centimeters of urobilin was excreted.

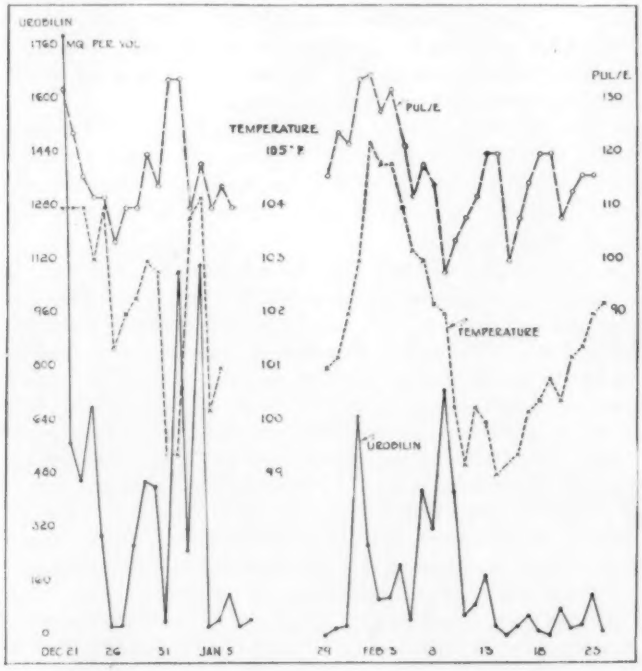

Chart 8.—Fluctuation in the urobilinuria in case 6.

However, the patient was confined to bed for the entire day on July 8, and, for these twenty-four hours only, slight traces of urobilin appeared in the urine. The tonsils were removed on July 9. On the same day the urobilinuria rapidly rose to 277 mg. and remained high for three days. A severe precordial pain distressed the patient for two days following the tonsillectomy. On July 15, the child was permitted out of bed for one-half day. The urobilinuria rose to 306 mg. for the twenty-four hours, but after the patient had rested in bed during the following day, it decreased to 11 mg. July 17 was an unusually hot and humid day, during which the child was irritable and restless, and complained of a severe pain in the chest. A precipitate rise in the excretion of urobilin to 257 mg. was noted for this day. On July 21, 24, 25, 26 and 27 the patient was permitted out of bed each day for periods varying from two to four hours. However, during July 19, 20, 22, 28,

29 and 30 the child lay quietly in bed. The absence of urobilinuria on the days the child was confined to bed forms a significant contrast to the amounts of urobilin that was excreted on the days part of which were spent out of bed. The fact that a definite hyperurobilinuria appeared on the days of activity in the absence of clinical symptoms of congestive failure, suggests that urobilinuria may be a valuable gage of the activities of the child with a cardiac condition.

Although the curve for the pulse rate showed excursions in a direction similar to that of the urobilinuria, the fluctuations were far less precipitate. On July 16, the maximum pulse rate was 128, and eleven days were required to diminish this rate to 96. However, the excretion of urobilin fell from 307 mg. per hundred cubic centimeters on July 16 to mere traces within forty-eight hours. Moreover, during the subsequent nine days, physicial exertion provoked a distinct rise in

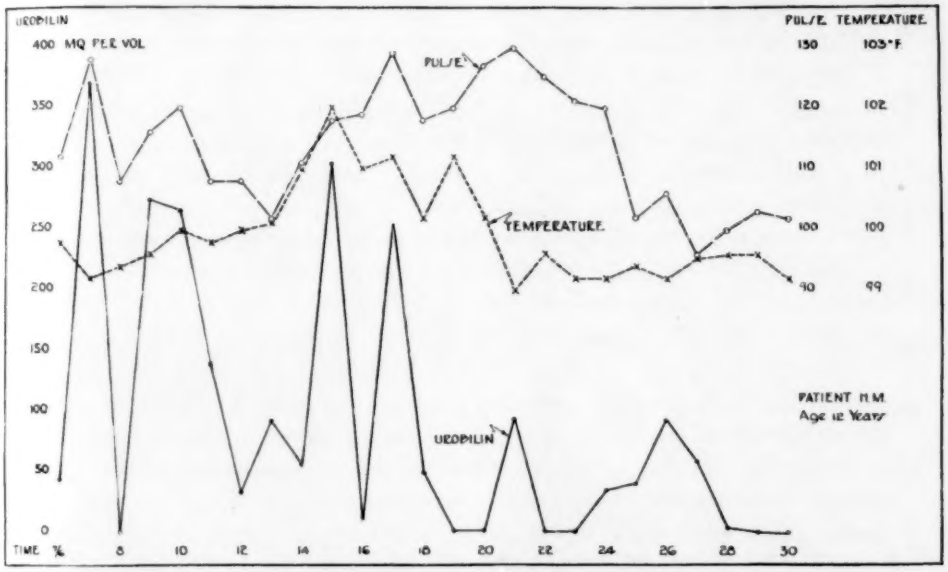

Chart 9.—Fluctuation in the urobilinuria in case 7.

urobilinuria, which far exceeded physiologic variations, but changes in the pulse rate were not greater than the diurnal fluctuations that are expected in a normal person.

COMMENT

The question arose during the course of this investigation whether information of greater practical value could not be obtained from quantitative determinations of the urobilinuria at frequent intervals during the day rather than from one estimation of a mixed twenty-four hour collection. To answer this question, separate analyses were made of all specimens of urine voided throughout twenty-four hour periods by a number of patients maintained throughout the day on standard conditions. Chart 10 presents graphically the results obtained in three cases, and these results are typical of all other cases that have been

studied. These observations indicate that during the night and the early morning hours, the urobilinuria disappears or falls to a low level, but reaches its maximum during the day; in our patients it reached its maximum during the afternoon hours. Saillet[7] observed that the urobilin that was excreted during the eight hours of sleep was only one tenth of the total amount eliminated during twenty-four hours. The fall in nocturnal urobilinuria, Saillet ascribed to a slackening of the processes of digestion and metabolism. An exhaustive study of diurnal variations in urobilinuria was reported by Wilbur and Addis[8] who determined the amount of urobilin that was excreted within two-hour

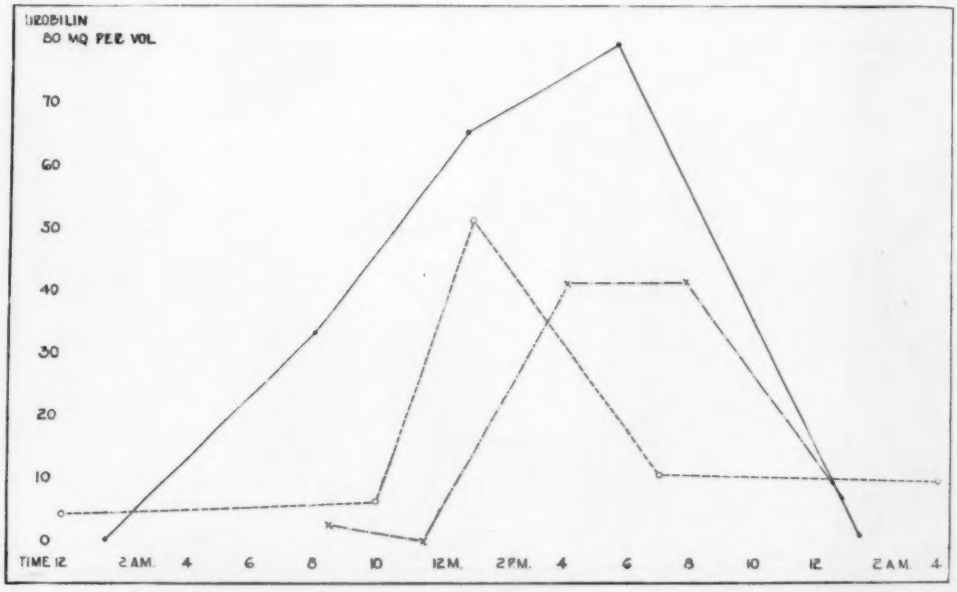

Chart 10.—Curves showing the fluctuation of urobilinuria in three typical cases.

intervals over a period of a month in a patient who had bronzed diabetes, maintained under standard conditions. A constant observation throughout this entire period was that during the hours of sleep, either day or night, the urobilinuria reached the minimum, but the maximum excretion was observed during the waking hours, the exact time of its occurrence was not constant, however. From a comparison of the amount of excretion of water, total solids, chlorides and urea with the amount of excretion of urobilin throughout the day, the suggestion

7. Saillet: Urobilin in Normal Urine, Rev. de méd. **17**:109, 1897; A New Urinary Pigment, Uroseine, Rev. méd. de la Suisse Rom. **17**:637, 1897.

8. Wilbur, R. L., and Addis, T.: Urobilin: Its Clinical Significance, Arch. Int. Med. **13**:235 (Feb.) 1914.

was made that observed fluctuations in excretion of urobilin were not due to variations in renal function, but possibly might be attributed to insufficiency of hepatic function during the hours of activity. Adler [9] reported a high excretion of urobilin during the morning in patients with a pathologic condition. Since there are such marked variations in urobilinuria at different periods of the day, it is evident that quantitative determinations of the urobilinuria in casual specimens of urine are of little value. Diurnal variations in excretion of urobilin cannot be interpreted as indicating an improvement or lack of improvement in cardiac function. We believe that the most dependable index of the urobilinuria is the total output during twenty-four hours.

McMaster and Rous [10] showed that in dogs and monkeys, over 80 per cent of the liver may be obstructed before bilirubinuria will result, and that in the dog more than 95 per cent of the organ must be occluded before jaundice occurs. However, in the experiments of Ellman and McMaster, urobilinuria regularly followed an obstruction of one third of the liver. Hence urobilinuria is a valuable index of the function of the liver in that it makes manifest a disturbance of mild degree in the liver which is not discernible in any other way. From these observations it can be inferred that the excretion of urobilin in the urine would more closely parallel the impairment of function of the liver consequent to cardiac decompensation than it would the bilirubin of the blood serum.

Bernheim [11] found that in forty-one cases of myocardial and chronic cardiovalvular disease, the icterus index varied directly with the degree of decompensation. In eleven cases that gave normal indexes there were no signs of decompensation. With slight evidences of cardiac failure, the index ranged from 7 to 9, and in seven cases with indexes of from 12.5 to 15, all but one case ended fatally within one to seven weeks.

Shattuck, Browne and Preston,[12] in studies of the icterus index of the blood serum in adults with cardiac disease, reported that this estimation of bilirubinemia promised some help in determining the degree of decompensation and prognosis in advanced cases of cardiac decompensation. However, the published data of the latter authors do not bear

9. Adler, A.: Urobilin, Deutsches Arch. f. klin. Med. **140**:302 (Nov. 2) 1922.

10. McMaster, P. D., and Rous, P.: The Biliary Obstruction Required to Produce Jaundice, J. Exper. Med. **33**:731, 1921.

11. Bernheim, A. R.: The Icterus Index (a Quantitative Estimation of Bilirubinemia): Aid in Diagnosis and Prognosis, J. A. M. A. **82**:291 (Jan. 26) 1924.

12. Shattuck, H. F.; Browne, J. C., and Preston, M.: Clinical Value of Some Recent Tests for Liver Function, Am. J. M. Sc. **170**:510, 1925.

out this conclusion, because they described cases in patients with slightly enlarged livers, but with normal figures for icterus index, although the prognosis was apparently poor. In two instances only of all the cases studied in this investigation did the icterus index of the blood serum exceed the normal limits of from 3 to 6. In one patient, L. F. (chart 6), an index of 10, with a positive indirect reaction to the van den Bergh test was obtained. This child left the hospital in an improved condition, and subsequently showed no symptoms of decompensation up to the time this article was written. Variations in icterus index of the blood serum of E. B. are graphically reported in chart 7, which demonstrates in a striking manner the relative influence of cardiac decompensation on bilirubinemia and urobilinuria. During the period of acute decompensation from October 12 to 18, the amount of excretion of urobilin rose from slight traces to 350 mg. per hundred cubic centimeters for twenty-four hours, but the icterus index was increased only from 10 to 11.5. A variation of 1.5 in icterus index is not outside the limits of experimental error and is insignificant. Furthermore, with the subsidence of symptoms of decompensation the urobilinuria promptly falls again to traces, but the icterus index is decreased only to 10 and persists at this level until death. However, three periods of hyperurobilinuria appear coincident with cardiac hypofunction in patients during days of unrestricted activities, and death is preceded by a rising urobilinuria. These variations in excretion of urobilin appear strikingly definite far exceeding the limits of technical error, and they form a marked contrast to the minor changes in icterus index. Blankenhorn [13] showed that urobilin is not a threshold substance and that renal impairment does not provoke a retention of urobilin. Wilbur and Addis demonstrated that the quantity of urobilin excreted is independent of the volume of urine voided. Hence the existence of diminished renal function, dependent on cardiac failure, does not detract from the practical value of urobilinuria, although the cardiac decompensation may be accompanied by passive congestion of the kidneys, or by a nephritis.

Clinically demonstrable enlargement of the liver, from one to four fingerbreadths below the costal margin, was found in the children with cardiac conditions with urobilinuria. It was not possible, however, to indicate any parallelism between the magnitude of the urobilinuria, or its fluctuations and enlargement of the liver. Cultures of the blood were made in only 20 per cent of the cases within the decompensated group, and these cultures yielded no bacterial growth after forty-eight hours of incubation.

13. Blankenhorn, M. A.: The Distribution of Bile in Certain Types of Jaundice, Arch. Int. Med. **21**:282 (Feb.) 1918.

St. Lawrence [14] stated that fever, i. e., body temperature by rectum above 99.6 F., practically always accompanies cardiac failure in children. The observations of this author indicate that at the onset of the attack of decompensation, a definite rise in temperature is noted, varying between 100 to 104 F., but as the cardiac failure progresses, the fever subsides, and often toward the end, the temperature may reach a daily peak only a fraction of a degree above 99.6 F. The temperature curves reported in the charts correspond closely with the observations of St. Lawrence. A definite increase in rectal temperature, from 102 to 104 F., is observed during the initial period of cardiac decompensation, coincident with a hyperurobilinuria. However, in the terminal stages of cardiac failure a decline in temperature is noted in A. B., E. L. and E. B. to approximately the normal level of 99.6 F., and in M. S., to a subnormal level. It is during these later stages of cardiac decompensation, particularly that a divergence of the urobilin curve from the temperature curve appears. In all instances, death was preceded by a rising urobilinuria, and in many cases by a declining temperature. Moreover, exercise provoked a rise in the excretion of urobilin in the children with poor cardiac compensation, but the exercise had no constant effect on body temperature. These observations suggest that the urobilinuria may be utilized more satisfactorily than variations on body temperature to grade the exercise or activities of the child with a cardiac condition.

SUMMARY

Urobilinuria was noted in 88 per cent of the children with congestive cardiac failure, in 27 per cent of the ambulatory children with cardiac disease, and in 4 per cent of the control cases.

The maximum concentration of urobilin in twenty-four hour specimens of urine from the decompensated group was twenty-five times greater than the maximum for the ambulatory group and sixty-five times greater than the maximum for the control cases.

Hyperurobilinuria followed tonsillectomies and myringotomies in 86 per cent of the children with congestive failure, and in 2 per cent of the control children of the group. Hyperurobilinuria after these surgical procedures was not observed in the children of the ambulatory group.

The quantity of urobilin in the urine of children with cardiac disease varies throughout the day. For the children whose activities were unrestricted in the babies' wards, the maximum amount that was excreted was noted in the afternoon hours between 12 m. and 6 p. m.

14. St. Lawrence, W.: The Problem of Exercise for Children with Heart Disease, J. A. M. A. **89**:2235 (Dec. 31) 1927.

The estimate of the prognostic value of urobilinuria in children with congestive cardiac failure was based on a comparison of the quantity of urobilin excreted during periods of twenty-four hours each with the clinical signs of cardiac failure throughout the period of hospitalization of twenty children.

Data for seven cases are presented graphically in this paper.

These observations suggest that urobilinuria is a valuable laboratory aid for the prognosis of the child with a congestive cardiac failure.

The urobilinuria should be considered as an important factor in determining the necessity of restricting the child's activities, or the advisability of surgical procedures such as transfusions and tonsillectomies.

INFLUENCE OF FEEDING ON CERTAIN ACIDS IN FECES OF INFANTS

IV. THE EFFECT OF AN EXCESS OF LACTOSE IN BREAST MILK AND IN WHOLE COW'S MILK ON THE EXCRETION OF VOLATILE ACIDS AND OF LACTIC ACID *

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While it is generally agreed that volatile and lactic acids in infants' stools are formed by the fermentation of lactose in the intestine, our previous studies [1] both with diluted cow's milk and with whole cow's milk have demonstrated that the amount of acid excreted is independent of the amount of lactose in the diet. Since the infants fed on whole cow's milk excreted as much as or even more volatile acid than the infants fed on breast milk, there must be a factor in cow's milk which facilitates the fermentation of lactose. In their clinical observations, Finkelstein and Meyer emphasized this point repeatedly; i. e., that carbohydrate is not harmful in itself, but only when given in a combination with cow's milk. They believe that the whey of cow's milk is the factor which acts as a catalyzer to intestinal fermentation. If the combination of carbohydrate and whole cow's milk is injurious to the whole intestinal tract, the addition of an excess of lactose to breast milk and to whole cow's milk should show striking differences in acid excretion. The investigation reported was therefore undertaken.

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* From the Otto Baer fund for Clinical Research, the Sarah Morris Hospital for Children, and the Nelson Morris Institute for Medical Research of the Michael Reese Hospital.

1. Gerstley, J. R.; Wang, C. C.; Boyden, R. E., and Wood, A. A.: The Influence of Feeding on Certain Acids in the Feces of Infants: I. A Comparison of the Effects of Breast Milk and Modified Cow's Milk on the Excretion of Volatile Acids, Am. J. Dis. Child. **35**:580 (April) 1928; II. The Effect of an Excess of Lactose in Breast Milk and in Modified Cow's Milk, with Observations on a Parenteral Infection, ibid. **36**:289 (Aug.) 1928. Gerstley, J. R.; Wang, C. C., and Wood, A. A.: The Influence of Feeding on Certain Acids in the Feces of Infants: III. A Comparison of the Effects of Breast Milk and Whole Cow's Milk on the Excretion of Volatile Acids and of Lactic Acid, Am. J. Dis. Child. **39**:487 (March) 1930.

We have already discussed the effect of an excess of lactose in breast milk. It was found that formic and acetic acids were unaffected. Propionic acid did not appear, and the total output of volatile acid as well as the total titratable acidity was usually within the limits of normal, occasionally slightly increased. There was little influence on the weight of the stool. Since then, the experiments have been repeated and, in addition, the amounts of lactic acid and the hydrogen ion concentration determined.

TABLE 1.—*Acid Excretion After Feeding*

			Total Cc. of Tenth-Normal Acid per 24 Hours					Cc. of Tenth-Normal Acid per 24 Hours per Kilogram of Body Weight				
Age	Infant	p_H	Formic	Acetic	Total Volatile	Total Titratable	Lactic	Formic	Acetic	Total Volatile	Total Titratable	Lactic
			Breast Milk Plus 3% Lactose									
7	F	....	45.9	63.8	109.7	74.4	84.5	11.7	16.3	28.0	18.9	21.5
			Breast Milk Plus 6% Lactose									
7	A	....	37.8	14.5	52.3	.....	.....	11.0	4.2	15.2	.....	.....
7	A	....	25.2	4.3	30.0	.....	.....	7.4	1.4	8.8	.....	.....
8	A	....	19.3	4.7	24.0	.....	.....	5.2	1.3	6.5	.....	.....
8	A	....	34.6	11.3	45.9	.....	.....	9.3	3.0	12.3	.....	.....
9	A	....	27.1	24.0	51.1	.....	.....	6.9	6.1	13.0	.....	.....
9	A	....	41.0	48.0	89.0	.....	.....	10.5	12.3	22.8	.....	.....
15	A	....	35.7	38.6	74.3	.....	.....	6.7	7.3	14.0	.....	.....
15	A	....	53.2	35.6	88.8	.....	.....	10.0	6.7	16.7	.....	.....
16	A	....	23.9	23.4	47.3	.....	.....	4.3	4.2	8.5	.....	.....
			Breast Milk Plus 8% Lactose									
8	D	....	42.3	82.5	124.8	64.7	98.4	9.9	19.4	29.3	15.2	23.1
9	D	....	40.1	58.3	98.4	47.7	118.8	9.0	13.1	22.1	10.8	26.9
9	D	....	40.9	14.1	55.5	39.9	48.5	9.2	3.2	12.4	8.9	10.8
			Breast Milk Plus 10% Lactose									
11	B	....	40.4	47.5	87.9	97.3	.....	8.8	10.3	19.1	21.1	.....
11	B	....	30.7	23.7	54.4	46.0	.....	6.7	5.1	11.8	10.0	.....
12	B	....	18.0	25.7	43.7	25.5	.....	3.6	5.2	8.8	5.1	.....
12	B	....	33.8	79.7	113.5	99.5	.....	6.8	6.1	12.9	20.1	.....
13	B	....	17.8	28.2	46.0	36.3	.....	3.5	5.5	9.0	7.0	.....
13	B	....	30.9	32.1	63.0	40.7	.....	6.0	6.2	12.2	7.9	.....
			Breast Milk Plus 12% Lactose									
12	M	4.48	8.7	60.9	69.7	84.3	56.8	1.7	12.0	13.7	16.6	11.2
12	M	4.76	12.3	61.6	73.9	99.9	42.8	2.4	11.9	14.3	19.4	8.3
17	A	...	39.0	37.4	76.4	.....	.....	6.7	6.5	13.2	.....	.....
17	A	....	52.7	54.8	107.5	.....	.....	9.1	9.5	18.6	.....	.....
17	C	....	18.4	51.6	70.0	68.1	.....	3.3	9.2	12.6	12.2	.....
17	C	....	5.9	13.5	19.4	9.3	.....	1.1	2.4	3.5	1.7	.....
18	A	....	47.3	58.0	105.3	.....	.....	7.9	9.6	17.5	.....	.....
18	A	....	42.7	58.7	101.4	.....	.....	7.1	9.8	16.9	.....	.....
18	C	....	24.4	58.0	82.5	83.3	.....	4.3	10.2	14.5	14.6	.....
19	A	....	40.7	22.2	62.9	.....	.....	6.6	3.6	10.2	.....	.....
19	A	....	29.6	36.4	66.0	.....	.....	4.8	5.9	10.7	.....	.....

The same technic of feeding, collecting specimens and chemical analysis as has been previously reported was employed in this series of experiments. Infants were fed on breast milk, breast milk with an excess of lactose, whole cow's milk and whole cow's milk with an excess of lactose. In some experiments, the order of feeding was reversed.

COMMENT

In table 1, we have grouped all the determinations on breast milk plus varying amounts of lactose. For simplicity the result of one

typical experiment is shown graphically in chart 1. Unquestionably, the addition of lactose to breast milk causes some increase in the excretion of lactic acid. When the feedings of breast milk plus 12 per cent of lactose were discontinued and the infant was given unmodified breast milk, the number of stools and their weight diminished. Clinically, lactose added to breast milk exerts a mildly laxative action. In this case chemical and clinical observations agree.

Table 2 gives the results of whole cow's milk, plus 3 and 12 per cent of lactose.

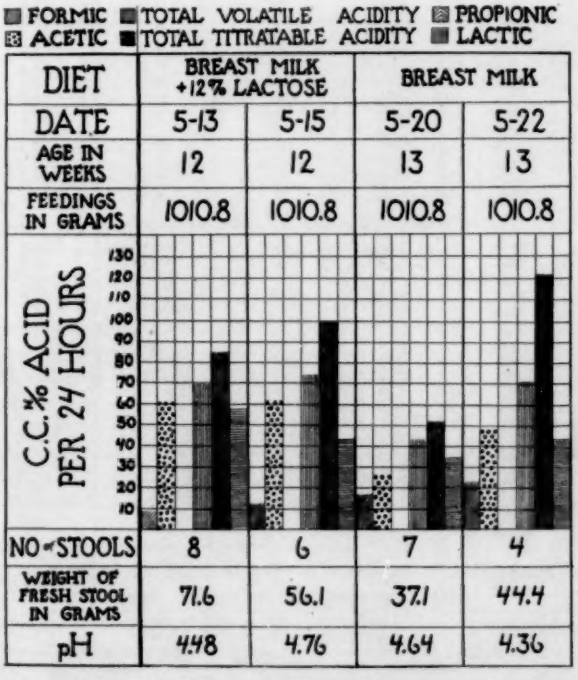


Chart 1.—The influence of the addition of 12 per cent of lactose to breast milk on the quantity of feces, the frequency of defecation, the excretion of volatile, total titrable and lactic acids and on the hydrogen ion concentration.

Table 3 shows the effects of all the feedings used on the hydrogen ion concentration of the feces.

The effect of 3 per cent of lactose added to whole cow's milk, as shown in charts 2 and 3, was quite variable. Chart 2 gives the results of an infant making an extremely rapid gain in recovering from a parenteral infection, while chart 3 gives those of an infant gaining normally and in good health. In general, the effect of an addition of 3 per cent of lactose tended to cause an increase in acetic acid and a diminution in propionic. There was no great change in total volatile acidity while the lactic and the total titratable acids increased.

The addition of 12 per cent of lactose to whole cow's milk resulted in relatively little change in the intestinal reactions. To our surprise, the acetic acid was oftener lower than when infants were given whole

TABLE 2.—*Acid Excretion and Hydrogen Ion Concentration After Feeding*

Age	Infant	p_H	Total Cc. of Tenth-Normal Acid per 24 Hours: Formic	Acetic	Propionic	Total Volatile	Total Titratable	Lactic	Cc. of Tenth-Normal Acid per 24 Hours per Kilogram of Body Weight: Formic	Acetic	Propionic	Total Volatile	Total Titratable	Lactic
Whole Cow's Milk Plus 3% Lactose														
9	J	6.26	....	73.8	13.8	87.6	71.5	71.1	....	11.0	2.0	13.0	10.6	10.6
9	J	5.90	....	155.0	5.5	160.6	139.8	85.5	....	22.9	0.8	23.7	20.6	12.6
10	J	5.95	....	87.0	17.4	104.4	70.9	57.7	....	12.5	2.5	15.0	10.2	8.3
10	J	5.56	31.7	97.4	....	128.7	117.0	131.2	4.5	13.8	...	18.3	16.5	18.5
11	G	5.48	58.7	49.1	16.0	123.8	187.4	178.5	13.7	11.5	3.7	28.9	43.8	41.7
12	G	5.80	5.2	81.4	....	86.6	86.4	45.4	1.1	18.0	...	19.1	19.1	10.0
12	G	5.51	....	59.3	2.1	61.4	37.6	23.1	....	13.3	0.4	13.7	8.4	5.2
14	H	6.88	....	103.7	43.7	147.4	29.8	13.3	....	18.6	7.8	26.4	5.3	2.4
14	H	7.53	....	43.5	27.5	71.0	6.9	4.2	....	7.6	4.8	12.4	1.2	0.7
15	F	....	....	130.5	7.7	138.3	74.2	60.5	....	22.1	1.3	23.4	12.6	10.3
15	F	....	....	141.6	27.5	169.1	137.8	93.8	....	24.1	4.7	28.8	23.5	16.0
17	F	....	....	70.2	2.0	72.2	90.8	27.5	....	12.1	0.3	12.4	15.8	4.8
17	F	....	....	86.5	0.8	87.2	61.8	31.1	....	14.7	0.1	14.8	10.5	5.3
Whole Cow's Milk Plus 12% Lactose														
12	J	5.31	....	70.8	....	70.8	82.0	88.0	....	9.2	...	9.2	10.6	11.4
12	J	5.15	15.1	76.0	....	91.2	136.8	96.2	1.9	9.8	...	11.7	17.7	12.5
13	J	5.20	....	73.9	0.1	74.0	66.1	47.6	....	9.2	...	9.2	8.2	5.9
13	J	4.96	....	88.8	....	88.8	129.7	57.6	....	10.9	...	10.9	16.0	7.1
13	G	5.37	....	68.4	1.2	69.6	59.0	37.0	....	13.5	0.2	13.7	11.6	7.3
14	J	5.25	....	55.9	2.3	58.3	75.0	74.6	....	6.7	0.3	7.0	9.0	8.9
14	G	5.41	....	60.8	7.1	67.9	57.5	23.6	....	11.6	1.4	13.0	10.9	4.3
14	G	5.26	....	58.2	3.5	61.7	54.8	25.9	....	10.7	0.6	11.2	10.1	4.8
15	G	5.05	....	116.9	3.1	120.0	242.7	83.3	....	21.4	0.6	22.0	44.4	15.1
15	G	6.59	....	27.2	5.8	33.0	15.5	8.7	....	4.9	1.0	5.9	2.8	1.6
15	H	6.12	....	109.4	9.2	118.6	38.2	40.7	....	18.2	1.5	19.7	6.4	6.8
15	H	6.73	....	69.0	15.7	84.7	15.5	4.5	....	11.2	2.5	13.7	2.5	0.7
19	F	....	67.6	102.6	....	170.2	134.8	112.5	10.5	15.9	...	26.4	20.9	17.4
19	F	....	77.8	9.8	27.0	114.6	50.0	52.5	11.9	1.5	4.1	17.5	7.6	8.0
20	F	....	....	73.3	8.5	81.8	40.9	26.6	....	11.0	1.3	12.3	6.2	4.0
20	F	....	....	142.2	1.2	143.5	55.6	31.6	....	21.2	1.8	23.0	8.3	4.7

TABLE 3.—*Hydrogen Ion Concentration in the Stools of the Infants on the Various Diets Arranged According to Age*

Breast Milk	Breast Milk Plus Lactose	Whole Cow's Milk	Cow's Milk Plus 3% Lactose	Cow's Milk Plus 12% Lactose
4.54	4.48	5.59	6.23	5.31
4.71	4.76	5.26	5.90	5.15
4.70	....	7.16	5.95	5.37
4.58	....	7.31	5.95	5.20
4.64	....	8.05	5.48	4.98
4.36	....	7.98	5.80	5.41
....	....	6.97	5.51	5.26
....	....	6.44	6.88	5.25
....	....	6.32	7.53	5.05
....	....	5.95	....	6.59
....	....	5.58	....	6.12
....	....	6.90	....	6.73
....	....	6.44	....	5.66
....	....	6.37	....	6.37
Averages 4.59	4.62	6.59	6.14	5.60

cow's milk with 3 per cent of lactose and at about the same level as or less than when they were given whole cow's milk alone. Formic and propionic acids both remained low. Lactic acid, too, while often in greater quantity than when whole cow's milk was used, was usually less

than when whole cow's milk with 3 per cent of lactose was used. The total volatile acid excretion became less than with the 3 per cent of lactose, and the total titratable acidity remained about the same. Figures expressed in per kilogram of body weight gave much the same results.

The most striking effect of the addition of lactose to whole cow's milk was in the change of the hydrogen ion concentration. A marked lowering of the p_H followed the addition of 3 per cent. The addition of 12 per cent caused a further lowering. The reaction practically never

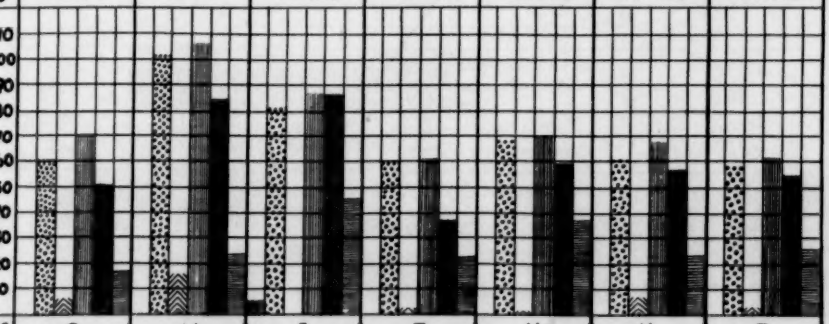
DIET	COW'S MILK		COW'S MILK +3% LACTOSE		COW'S MILK +12% LACTOSE		
DATE	5-29	6-1	6-12	6-14	6-22	6-26	6-28
AGE IN WEEKS	10	10	12	12	13	14	14
FEEDINGS IN GRAMS	929.6	917.4	870.8	964.1	870.8	777.5	762.0
C.C.% ACID PER 24 HOURS							
NO. STOOLS	9	11	8	3	4	4	5
WEIGHT OF FRESH STOOL IN GRAMS	68.3	145.7	92.0	71.9	69.7	63.3	69.6
pH	6.44	6.37	5.80	5.51	5.37	5.41	5.26

Chart 2.—The influence of the addition of 3 and of 12 per cent lactose to whole cow's milk on the quantity of feces, the frequency of defecation, the excretion of volatile, total titratable and lactic acids and on the hydrogen ion concentration in an infant recovering from a parenteral infection.

reached the acidity of breast milk. In general, the gradual increase of lactose in whole cow's milk brings about an acidity approaching that of the stool from infants fed on breast milk, but never identical with it. The suggestion is that these changed relations may be due to the shifting ratios of carbohydrate and protein.

The relation of these studies to the prevailing theories of infant feeding will be discussed in a later publication. Suffice it to say that in no case did severe diarrhea develop from the addition of 12 per cent of lactose to whole cow's milk, while occasionally there were fewer

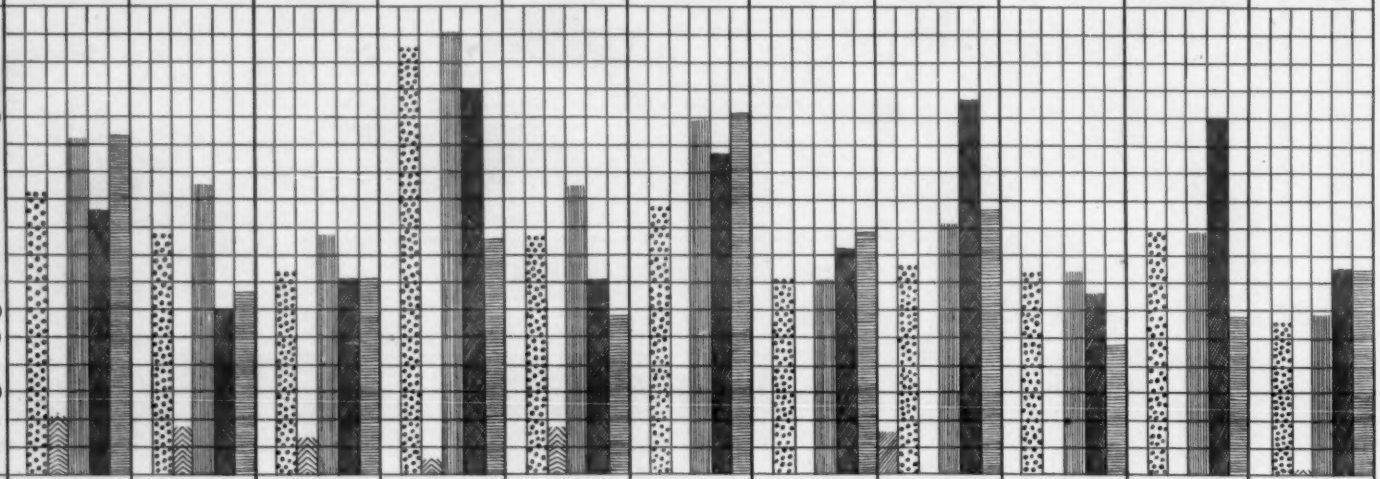
DIET	WHOLE COW'S MILK		WHOLE COW'S MILK +3% LACTOSE				WHOLE COW'S MILK +12 % LACTOSE				
DATE	10-29	10-31	11-12	11-14	11-19	11-21	12-3	12-5	12-10	12-12	12-17
AGE IN WEEKS	7	7	9	9	10	10	12	12	13	13	14
FEEDINGS IN GRAMS	746.4	762.0	870.8	870.8	870.8	855.2	870.8	855.2	870.8	870.8	870.8
C.C.% ACID PER 24 HOURS											
NO of STOOLS	12	9	8	9	9	11	10	12	4	9	9
WEIGHT OF FRESH STOOL IN GRAMS	348.7	173.5	178.1	209.8	105.1	151.7	125.7	111.3	83.7	121.0	70.5
pH	6.97	6.44	6.26	5.90	5.95	5.56	5.31	5.15	5.20	4.98	5.25

Chart 3.—The influence of the addition of 3 and of 12 per cent lactose to whole cow's milk on the quantity of feces, the frequency of defecation, the excretion of volatile, total titratable and lactic acids and on the hydrogen ion concentration in a normal infant.

stools than on cow's milk alone. These observations plus the diminished rather than the increased acid output indicate that this combination given to a well infant does not cause diarrhea of primary intestinal origin. It is interesting to note that even with a high lactose content the p_H never reached the acidity of the stool from infants fed on breast milk. How much lactose would be required and whether such an amount could be given to an infant is a question for further study.

CONCLUSIONS

The addition of 3 per cent of lactose to whole cow's milk causes an increase in the output of volatile acid, of lactic acid and in the total titratable acidity. The addition of 12 per cent of lactose shifted the output of the volatile acid toward that of cow's milk and of lactic and total titratable acids toward breast milk. These changes were never sufficient to cause primary intestinal diarrhea.

Three per cent of lactose often showed little effect on the weight of the stool when added to cow's milk. Twelve per cent of lactose often produced great change, reducing the weight to approximately that of the stool from infants fed on breast milk.

The addition of lactose to breast milk causes little change in the hydrogen ion concentration. The addition of lactose to cow's milk results in an increased acidity of infants' stools, the most marked change taking place when 3 per cent of lactose is added to the milk and a much smaller change in proportion when 12 per cent is added. Varying of the amounts of carbohydrate added to cow's milk never resulted in changes of acidity such as occurred when the diet was changed from breast milk to whole cow's milk.

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THE RESPIRATORY METABOLISM IN INFANCY AND IN CHILDHOOD

XI. THE RESPIRATORY EXCHANGE IN MARASMUS: EFFECT OF MUSCULAR ACTIVITY *

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A few workers [1] have assigned the major rôle in the pathogenesis of infantile marasmus to an abnormal energy metabolism. Studies reported in the three preceding papers of the present series [2] do not support this view. These studies demonstrated that the basal metabolism, the energy metabolism following the ingestion of the principal foodstuffs, protein, carbohydrate and fat, and the intermediary metabolism of the latter food factors were all essentially normal even in severe grades of infantile marasmus. The remaining derangement of energy metabolism, which may be a contributing factor in maintaining the marasmic state, is an abnormally elevated metabolic response to muscular activity. This phase of the subject will be considered in the present communication.

It must be emphasized at the outset that the evaluation of the muscular efficiency of infants is at best a rough approximation, since accurate methods for quantitatively estimating mechanical work in these subjects are not at present available. If, however, the marasmic infant expends relatively more heat than the normal infant during exercise (crying) of similar duration and of approximately similar intensity, one

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* From the Department of Pediatrics, Cornell University Medical College, and the New York Nursery and Child's Hospital.

1. Marfan, A. B.: Traité de l'allaitement, Paris, 1920, p. 621. Utheim, K.: Advanced Chronic Nutritional Disturbances in Infancy, J. Metab. Research **1**: 803, 1922.

2. Levine, S. Z.; Wilson, J. R., and Gottschall, G.: The Respiratory Metabolism in Infancy and in Childhood: VIII. The Respiratory Exchange in Marasmus: Basal Metabolism, Am. J. Dis. Child **35**:615 (April) 1928. Wilson, J. R.; Levine, S. Z., and Gottschall, G.: The Respiratory Metabolism in Infancy and in Childhood: IX. Carbohydrate Metabolism of Normal and of Marasmic Infants with and without the Administration of Insulin, ibid. **36**:470 (Sept.) 1928. Levine, S. Z.; Wilson, J. R., and Gottschall, G.: The Respiratory Metabolism in Infancy and in Childhood: X. Specific Dynamic Action of Food in Normal and in Marasmic Infants, ibid. **36**:740 (Oct.) 1928.

would be justified in concluding that, regardless of the actual work achieved, the excessive dissipation of heat during activity might be a contributing factor in marasmus.

Another possibility exists. Even though the energy metabolism may not be abnormally elevated during activity, the marasmic infant may spend more of his day in crying and less in sleep than the normal infant. Such prolonged activity even with normal efficiency would also result in the excessive dissipation, as waste heat, of the potential energy of the ingested food and thus render it unavailable for purposes of maintenance and growth.

The present work was undertaken to determine whether either or both of these possibilities are factors in maintaining a condition of infantile marasmus.

Rubner and Heubner [3] were the first to estimate the effect of crying on the heat production of infants. Their results are not comparable with those of more recent studies because of differences in technic. These and more recent studies have been reviewed by Murlin.[4]

Howland,[5] while working in Dr. Lusk's laboratory, demonstrated increases of 18 and 39 per cent in the heat output of two infants aged 3 and 7 months in response to crying.

Murlin reported the following results:[6] A normal infant, aged 2 months and weighing 5.7 Kg., lost 11.89 calories an hour during sleep and 17.45 calories an hour when crying approximately two thirds of the experimental period. A second infant, 3 months old, weighing 4.14 Kg., lost 10.75 calories an hour during sleep. The heat output of this infant rose to 12.04 calories an hour when he fretted and cried for twenty out of a total of sixty minutes. Calculated on the basis of 100 per cent of crying time, the basal heat production rose 71 per cent in the first and 36 per cent in the second instance. These two cases are recorded in table 3.

Benedict and Talbot [7] reported the heat production of a group of newly born infants while at rest and during activity. The increase of the maximum over the minimum heat output reached the high figure of 211 per cent in one instance and averaged 65 per cent for the entire group. These authors give no figures for the duration of activity.

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3. Rubner, M., and Heubner, O.: Die künstliche Ernährung eines normalen und eines atrophischen Säuglings, Ztschr. f. Biol. **38**:315, 1899.

4. Murlin, J. R., in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, vol. 1, p. 521, 1923.

5. Howland, J.: Der Chemismus und Energieumsatz bei schlafenden Kindern. Ztschr. f. physiol. **74**:1, 1911.

6. Murlin, in Abt's Pediatrics, p. 660.

7. Benedict, F. G., and Talbot, F. B.: Physiology of the Newborn, Pub. 233, Carnegie Institution of Washington, 1915.

Murlin, Conklin and Marsh,[8] in a recent study of newly born infants, calculated that the basal metabolism was approximately doubled by crying. Despite some wide variations in individual infants, they believe that the metabolic response of these subjects to crying is, in general, fairly consistent.

METHODS

Studies with the Respiration Chamber.—A description of the metabolism ward and of the apparatus, the calculation of results and the technical procedures appeared in earlier papers of this series.[9] Frequent alcohol checks were made to control the reliability of the observations. These data were reported in papers VI and VII.[10]

Each infant was studied in at least two observations of from one to two hours' duration, one during sleep and one during activity. In the latter, the proportion of time occupied in crying was noted by an observer at the window of the respiration chamber and checked by a graphic record of activity.[11] These observations were terminated only after the subject had fallen asleep and conditions in the chamber had resumed a steady state. The time interval between the cessation of crying and the termination of observations varied between twenty and forty minutes. The reasons for these precautions and possible sources of error were briefly discussed in paper V.[12] Basal and active experiments were made either on the same day or at intervals of not more than four days and were invariably preceded by identical meals.

Observations of the Daily Activity.—The daily behavior of six infants in varying states of nutrition was accurately recorded by one of us (M. K.) or an assistant for a total of thirty-three days. Each

8. Murlin, J. R.; Conklin, R. E., and Marsh, M. E.: Energy Metabolism of Normal New-Born Babies with Special Reference to the Influence of Food and of Crying, Am. J. Dis. Child. **29**:1 (Jan.) 1925.

9. Levine, S. Z.; Wilson, J. R., and Rivkin, H.: The Respiratory Metabolism in Infancy and in Childhood: I. Basal Metabolism of Children, Am. J. Dis. Child. **31**:323 (March) 1926. Wilson, J. R.; Levine, S. Z., and Rivkin, H.: The Respiratory Metabolism in Infancy and in Childhood: II. Ketosis and the Respiratory Exchange in Children, ibid. **31**:335 (March) 1926.

10. Levine, S. Z.; Wilson, J. R.; Berliner, F., and Rivkin, H.: The Respiratory Metabolism in Infancy and in Childhood: VI. The Specific Dynamic Action of Food in Normal Infants, Am. J. Dis. Child. **33**:722 (May) 1927. Levine, S. Z., and Wilson, J. R.: The Respiratory Metabolism in Infancy and in Childhood: VII. Elimination of Water Through the Skin and Respiratory Passages of Infants, ibid. **35**:54 (Jan.) 1928.

11. Levine, Wilson and Rivkin (footnote 9, first reference).

12. Wilson, J. R.; Levine, S. Z.; Rivkin, H., and Berliner, F.: The Respiratory Metabolism in Infancy and in Childhood: V. Effect of Moderate Exercise on the Respiratory Quotient: Study of Four Children, Am. J. Dis. Child. **33**: 618 (April) 1927.

infant was under constant observation throughout the twenty-four hours of each day for from three to six successive days. Only one subject was observed at a time.

Each twenty-four hour period was partitioned into periods of sleep and wakefulness, and the latter was further subdivided into periods of quiet, crying, feeding and restlessness (table 4). The onset and termination of crying were spontaneous in all instances. Except during feeding and for from two to four minutes thereafter when the infants were held up by the nurse, they remained prone in their cribs or on the metabolism frame.

TABLE 1.—*Summary of Nutritional Condition of Subjects*

Case *	Experiment	Age, Months	Date	Weight, Kg.	Average Weight for Age, Kg.†	Percentage Underweight for Age
		Calorimeter Observations				
3	42	11	11/26/26	7.80	9.32	—16
12	79	5¼	4/18/28	5.98	7.36	—19
	78	5¼	4/16/28	5.91	7.36	—20
11	74	7½	1/16/23	6.54	8.31	—21
	75	7½	1/20/28	6.59	8.31	—21
5	58	7½	1/28/27	6.44	8.31	—23
5	32	4½	11/ 1/26	3.71	6.97	—47
	30	4½	10/27/26	3.67	6.97	—47
4	13	5	2/26/26	3.64	7.24	—50
	14	5	2/27/26	3.64	7.24	—50
5	26	4	10/18/26	3.21	6.69	—52
		Observations of Daily Activity				
14	..	8½	5/18/28	7.90	8.64	—8
12	..	5½	4/25/28	6.22	7.52	—17
11	..	8½	2/11/28	6.80	8.60	—21
11	..	8¼	2/ 6/28	6.70	8.55	—22
5	..	7⅓	1/25/27	6.39	8.26	—23
13	..	3½	4/ 2/28	3.95	6.38	—38
5	..	5½	11/26/26	4.32	7.45	—42
5	..	4¼	10/21/26	3.41	6.84	—50
10	..	2⅓	2/17/27	2.70	6.83	—60

* All subjects were males.

† Woodbury tables Pub. 87, Children's Bureau, U. S. Dept. of Labor, 1921, p. 107.

Although this method of observation by inspection is not entirely satisfactory without graphic records, great care was taken to record the behavior as accurately as possible, and since the same routine was followed in all instances the method seems acceptable for comparative purposes. The partitioning of the different periods is probably no more than a close approximation.

REPORT OF CASES

The subjects included eight infants from 2 to 11 months of age and from 8 to 60 per cent underweight for age. The emaciated subjects presented typical cases of infantile marasmus. A summary of the nutritional condition of all the subjects at the time of the observations appears in table 1.

Three of the subjects were studied in the respiration chamber and observed throughout the twenty-four hours (cases 5, 11 and 12); two infants were studied by the first method only (cases 3 and 4), and the three remaining infants were studied solely by the second procedure (cases 10, 13 and 14). All subjects were free from parenteral infections and alimentary disturbances during observations. They were afebrile with one exception (case 5, experiment 59, temperature 100.5 F.).

CASE 3.—G. S., a colored boy, born Jan. 4, 1926, was admitted to the hospital on September 24 for placement by the Foster Home Department. The past and family histories were unobtainable. He weighed 14 pounds, 13 ounces (6,732 Gm.) on admission. On examination he seemed fairly well developed and in apparent health. When observed in the respiration chamber on November 26, he was 11 months old and weighed 17 pounds, 2 ounces (7,783 Gm.). The Wassermann and tuberculin tests were negative.

CASE 12.—K. F., a colored boy, born Nov. 7, 1927, entered the hospital at the age of 4 months, because of an infection of the upper respiratory tract and mild diarrhea. The past and family histories were not obtainable, as the patient was a foundling. He weighed 13 pounds, 2 ounces (5,965 Gm.). Recovery was uneventful, and he was discharged at the end of three weeks (March 30), weighing 12 pounds, 13 ounces (5,823 Gm.).

The child was readmitted April 9, with a very mild infection of the upper respiratory tract and because of failure to gain weight. He weighed 13 pounds, 6 ounces (6,079 Gm.). The acute infection subsided promptly, but his weight remained practically stationary. When discharged on June 9, he was 7 months old and weighed 14 pounds, 2 ounces (6,420 Gm.), having gained only 1 pound since his first admission three months previously. The Wassermann and tuberculin reactions were negative.

The patient was studied in the respiration chamber on April 16 and April 18, when he was approximately 20 per cent underweight for his age. His daily activity was recorded from April 25 to 27, inclusive.

CASE 11.—W. B., a colored boy, born May 29, 1927, was admitted on December 29 for placement by the Foster Home Department. The past and family histories were unknown. On examination he appeared well developed and, except for a moderate degree of undernutrition, in good health. He was well during his stay in the hospital and gained weight. When discharged on April 9, 1928, he was 10 months old and weighed 18 pounds, 5 ounces (8,323 Gm.). The Wassermann and tuberculin tests were negative.

The child was observed in the respiration chamber on January 16 and 20, when he was 22 per cent underweight for his age. His daily activity was also recorded for two periods of three days each, beginning on February 6 and February 11, respectively.

CASE 4.—J. B., a white boy, born Sept. 25, 1925, was admitted to the hospital at the age of 3 months because of marked malnutrition. The family history was unimportant. He had been born in the eighth month of gestation after an uneventful labor and weighed 5 pounds, 10 ounces (2,557 Gm.). Breast feedings had been discontinued at the age of 1 month and replaced by feedings of condensed milk, which he received up to the time of admission. On physical examination he appeared small and emaciated, weighing 6 pounds, ½ ounce (2,741 Gm.). The buttocks were excoriated, and there was bilateral otitis media. Both ear-

drums were subsequently incised, and pus was obtained. During the latter part of January, 1926, he suffered from a mild infection of the upper respiratory tract, but recovery was uneventful. The Wassermann and tuberculin reactions were negative.

When he was observed in the respiration chamber on Feb. 26 and 27, 1926, he was 5 months old and weighed 8 pounds (3,636 Gm.), being 50 per cent below the expected weight for his age.

CASE 14.—E. B., a white boy, was admitted on May 10, 1928, at the estimated age of 8 months. The past and family histories were not obtainable, as the patient was a foundling. He was well developed and well nourished, apparently in excellent health and weighed 17 pounds, 6 ounces (7,897 Gm.). He was finally placed in a foster home.

An accurate record was made of his daily behavior from May 18 through May 20, when he was 8 per cent below the average normal weight for his age.

CASE 13.—R. O'K., a white boy, was admitted to the hospital on March 10, 1928, at the estimated age of 2 months because of diarrhea and a mild degree of dehydration. The family and past histories prior to the onset of diarrhea were unknown. Examination on admission showed a well developed infant with moderate dehydration and a bilateral catarrhal otitis media. His weight was 8 pounds, 1 ounce (3,644 Gm.). During the twenty-four hours he lost almost 1 pound in weight, and dehydration was pronounced. The diarrhea subsided at the end of thirteen days, and he appeared markedly improved. Between April 2 and 6, during a period of stationary weight (8 pounds, 11 ounces, or 3,948 Gm.), his daily activity was accurately recorded. Shortly thereafter, the patient began to gain weight, and he was discharged from the hospital in good condition. The Wassermann and tuberculin tests were negative.

Cases 5 and 10 were briefly summarized in an earlier communication.[13]

RESULTS

Studies with the Respiration Chamber.—Details of the data obtained with the calorimeter appear in table 2. Measurements of the heat production during sleep and during exercise (crying) are placed adjacent to each other. Observations during sleep were not strictly basal in all instances, since the meals preceding the experiments were not always minimal, but in order to exclude as far as possible differences in the stimulating effect of food, the basal and the crying observations in each infant were invariably preceded by identical meals.

The duration of crying and the total length of the period varied in the different experiments. The crying time was therefore expressed in percentages of the total period (table 3, column 5), and the percentage increase above the basal production of heat with crying was similarly expressed (column 9). For purposes of comparison, the extra metabolism of crying was also calculated for each infant on the basis of 100 per cent of crying time (column 8) and expressed in terms of percentages above the respective basal levels (column 10).

13. Marfan (footnote 1, second reference).

TABLE 2.—*Calorimeter Data in Terms of Averages Per Hour*

Case	Experiment	Age Mos.	Weight Kg.	Height Cm.	Surface Area * Sq. M.	Duration of Observation Minutes	Carbon Dioxide Gm.	Oxygen Gm.	Respiratory Quotient	Urinary Nitrogen Gm. per Hour	Calories: Actual Per Hour	Calories: Per Kg. Per 24 Hours	Calories: Per Sq.M. Per Hour	Rectal Temp. Ave. ° F.	Comment †
3	42	11	7.80	71.0	0.417	81	9.13	7.38	0.900	0.3622	24.74	76.1	59.3	99.2	P. 41.5 Gm.; F. 7.8 Gm.;
	42		7.80	71.0	0.417	60	7.87	6.64	0.862	0.3622	21.97	67.6	52.7	99.2	C. 9.0 Gm.; Cal. 279.6
12	79	5¼	5.98		0.329 ‡	60	5.33	4.51	0.860	0.0774	15.25	61.2	46.4	98.3	P. 5.0 Gm.; F. 6.0 Gm.
	79		5.98		0.329	31	5.28	§		0.0774	15.10	60.6	45.9	98.3	C. 7.5 Gm.; Cal. 126.
12	78	5¼	5.91		0.327 ‡	75	5.54	4.98	0.808	0.0826	16.61	67.5	50.8	98.8	P. 5.0 Gm.; F. 6.0 Gm.;
	79		5.98		0.329	31	5.28	§		0.0774	15.10	60.6	45.9	98.3	C. 7.5 Gm.; Cal. 126.
11	75	7¾	6.59	68.8	0.364	20	7.35	6.01	0.890	0.1475	20.37	74.2	56.0	98.7	P. 4.4 Gm.; F. 5.3 Gm.
	74	7½	6.54	68.8	0.361	120	7.17	5.74	0.909	0.0823	19.66	72.1	54.5	99.9	C. 16.5 Gm.; Cal. 135.
5	58	7½	6.44	65.7	0.356	128	8.65	§		‡	24.20	90.2	68.5	99.8	P. 2.0 Gm.; F. 2.4 Gm.;
	59	7½	6.44	65.7	0.356	143	6.14	§		‡	17.97	67.0	50.5	100.5	C. 3.0 Gm.; Cal. 42.8; Urea 6 Gm.
5	30	4½	3.67	57.5	0.237	90	4.49	3.67	0.890	‡	12.47	81.6	52.6	98.8	P. 2.0 Gm.; F. 2.4 Gm.;
	32		3.71	57.5	0.239	150	4.12	3.37	0.891	0.0782	11.43	73.9	47.8	98.6	C. 3.0 Gm.; Cal. 42.8
4	14	5	3.64	55.5	0.236	105	4.95	4.20	0.858	0.1142	14.09	92.9	59.7	99.1	P. 12.8 Gm.; F. 3.4 Gm.;
	13		3.64	55.5	0.236	63	4.76	4.14	0.837		13.81	91.0	58.5	99.3	C. 4.0 Gm.; Cal. 100.5.
5	26	4	3.21	57.5	0.217	66	5.42	§		0.0611	13.88	103.8	64.0	99.2	Dextrose 35 Gm.; Cal. 129.5.
	26		3.21	57.5	0.217	60	4.00	§		0.0611	10.43	78.0	48.1	99.2	

* Surface area estimated from nomogram of R. R. Hannon, extended for infants and children according to the formula of Du Bois and Du Bois (Basal Metabolism in Health and Disease, ed. 2, Philadelphia, Lea & Febiger, 1927, p. 124).

† All meals consisted of whole milk dilutions and were given at least one and usually more hours preceding the onset of observation. P., F., C. and Cal. indicate protein, fat, carbohydrate and calories, respectively.

‡ Surface area of these infants was derived from the table of Benedict and Talbot (Metabolism and Growth from Birth to Puberty, Pub. 302, Carnegie Institution of Washington, 1921, p. 62.).

§ Oxygen specimens lost. Heat production was computed from carbon dioxide on the basis of respiratory quotients obtained in similar experiments.

‡ Specimen of urine lost. Heat production computed on assumption that 15 per cent of the total calories was derived from protein.

The extra metabolism per unit of crying time of the different subjects and of the same subject in different observations exhibited wide variations. No correlation was evident between the state of nutrition and the metabolic response to activity. The percentage rise in heat production per 100 per cent of crying time of the five subjects who were 23 per cent or less underweight for age ranged between 13 and 71 per cent; that for the three marasmic subjects who were 32 per cent or more underweight, between 7 and 129 per cent. The figures for children of the latter group fell below, within and above those for subjects of the former group (column 10, table 3). Furthermore, the same subject reacted differently to crying in different observations. One slightly undernourished infant (case 12) responded with increases

TABLE 3.—*The Effect of Crying on the Metabolism of Subjects in Different States of Nutrition*

		Activity			Calories per Hour			Percentage Increase in Metabolism	
		Crying Time							
Case	Underweight for Age, %	Total Period, Minutes	Minutes	% of Total Period	Basal Period, Cal.	Active Period, Cal.	Had Subject Cried 100% of Time, Cal.	Actual %	On Basis of 100% of Crying Time, %
*	— 7	61	40	66	11.89	17.45	20.23	47	71
3	—16	81	25	31	21.97	24.74	30.91	13	42
12	—20	60	5	8	15.10	15.25	16.99	1	13
		75	13	17	15.10	16.61	23.98	11	65
11	—22	120	10	8	19.66	20.37	28.54	4	50
5	—23	128	84	66	17.97	24.20	27.32	35	53
*	—32	60	20	33	10.75	12.04	14.62	12	36
5	—47	90	6	7	11.43	12.47	26.29	9	129
4	—50	105	30	29	13.81	14.09	14.78	2	7
5	—52	66	30	45	10.43	13.88	18.10	33	73

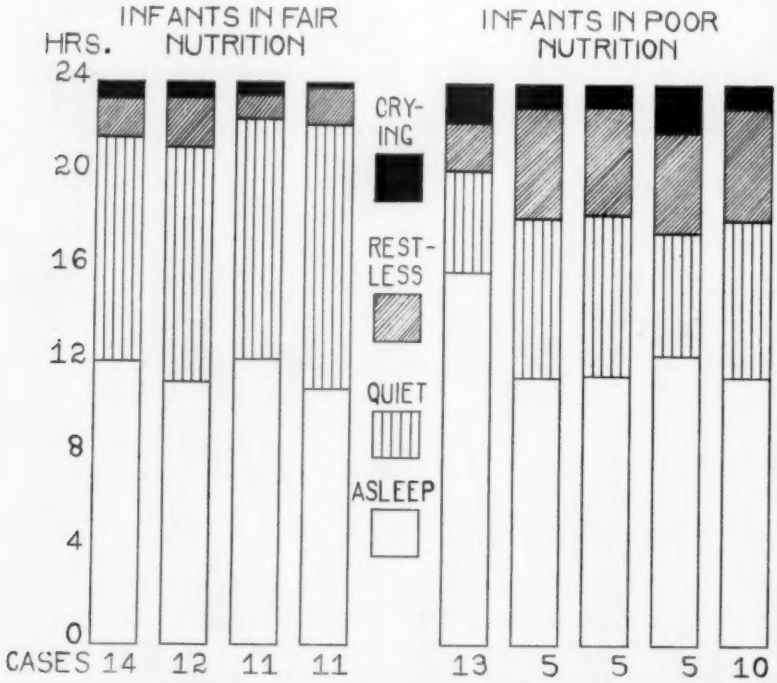
* Cases described by Murlin (footnote 4).

in metabolism of 13 and 65 per cent in two experiments on two successive days with no change in the nutritional condition. The extra metabolism of another infant (case 5) per 100 per cent of crying time was 73, 129 and 53 per cent above basal levels in three observations, and the augmentation of the metabolic rate was not proportional to the degree of undernutrition at the time of observation. These variations are to be expected, since the vigor and intensity of crying undoubtedly vary in different infants and even in the same subject at different times, except possibly in newly born infants in whom the character of crying may be more uniform. The latter conception, if true, may explain the comparatively uniform results obtained by Murlin and his co-workers [8] in newly born subjects.

The inconstant results obtained in these experiments afford no evidence of an abnormal expenditure of heat in marasmus in response to crying. It is of course conceivable that certain infants maintain their marasmic state because of excessive heat waste accompanying work, but conclusive proof of this conception must await more exact methods than are at present available for the quantitative measurement

of mechanical work in these subjects. The conclusion may be tentatively advanced until further observations are forthcoming that a disproportionate expenditure of waste heat accompanying activity is not a usual or important factor in maintaining the marasmic state.

Observations of the Daily Activity.—As previously mentioned, the possibility remains that emaciated infants are restless for a greater proportion of the day than well nourished infants and so dissipate an abnormally high quota of energy as waste heat. Data collected on



Comparison of the daily activity of infants in fair nutrition and of infants in poor nutrition during observations on from three to six successive days. The former subjects were from 8 to 22 per cent underweight for age; the latter, from 23 to 60 per cent underweight.

this phase of the subject are recorded in table 4 and in the accompanying chart.

The table and the chart show that with one exception (case 13) the proportion of the day occupied in sleep by all subjects was approximately the same, regardless of their nutritional condition, which varied from 8 to 60 per cent below the expected weight for age at the time the observations were made. Their daily sleeping time in periods of observation of from three to six days averaged between eleven and twelve and one-half hours (653 to 741 minutes) as against sixteen

hours (953 minutes) in case 13. The latter marasmic infant, however, was quiet when awake for a considerably shorter period (257 minutes) and cried for an appreciably longer period of the day (103 minutes) than the three normal infants, who averaged 620 minutes of quiet and 23 minutes of crying a day.

TABLE 4.—*The Daily Activity of Infants in Different States of Nutrition*

Case	Date	Age Months	Underweight for Age %	Awake				Sleeping Min.
				Crying Min.	Feeding Min.	Restless Min.	Quiet Min.	
14	5/18/28	8	8	10	24	119	646	641
	5/19/28			33	24	64	609	710
	5/20/28			18	25	95	477	825
	Average			20	24	93	577	725
12	4/25/27	5½	17	52	58	38	546	746
	4/26/27			31	79	80	583	667
	4/27/27			38	52	72	669	609
	Average			40	63	63	599	674
11	2/11/28	8½	21	0	21	0	681	738
	2/12/28			0	22	0	643	775
	2/13/28			82	21	115	547	675
	Average			27	21	38	623	729
11	2/ 6/28	8¼	22	16	21	111	637	655
	2/ 7/28			0	18	133	618	671
	2/ 8/28			0	13	0	793	634
	Average			5	17	81	683	653
13	4/ 2/28	3½	38	152	67	108	227	886
	4/ 3/28			105	40	66	246	983
	4/ 4/28			51	62	37	299	991
	Average			103	56	67	257	953
5	1/25/27	7½	23	75	125	125	412	703
	1/26/27			70	101	176	407	686
	1/27/27			44	54	268	423	651
	Average			63	93	190	414	680
5	11/26/26	5½	42	42	112	143	418	725
	11/27/26			92	100	198	364	687
	11/28/26			66	84	172	384	734
	11/29/26			12	104	153	471	700
	11/30/26			80	96	194	493	577
	Average			58	99	172	426	685
5	10/21/26	4¼	50	134	121	164	264	757
	10/22/26			101	83	210	327	719
	10/23/26			65	104	178	266	827
	10/24/26			225	97	161	282	675
	10/25/26			97	90	125	400	728
	10/26/26			120	88	117	373	742
	Average			124	97	159	319	741
10	2/17/27	2½	60	64	149	149	423	655
	2/18/27			64	116	190	400	670
	2/19/27			49	124	151	381	735
	2/20/27			55	128	157	370	730
	Average			58	129	162	394	698

If the daily periods of crying, restlessness and feeding on the one hand and those of sleep and quiet on the other are grouped into two main divisions of activity, as presented in the chart, one notes that the poorly nourished infants were invariably more active than the better

nourished ones. The figures in four observations on the latter group of three infants averaged twenty-one and one-third hours of quiet and two and two-third hours of activity, as compared to eighteen and one-half hours of quiet and five and one-half hours of activity a day in five observations on three infants of the former group. The average daily difference of nearly three hours is significant when one realizes that muscular activity exerts a more notable influence on the energy metabolism than all other physiologic factors combined. Furthermore, the portion of the active daily period spent in crying by the poorly nourished infants exceeded in every instance the crying time of the better nourished subjects.

It seems reasonable to conclude from these relatively few but careful observations that prolonged activity may contribute, in some instances at least, toward the maintenance of the marasmic state by rendering unavailable an abnormally high proportion of the potential energy of ingested foodstuffs for purposes of growth. In the absence of graphic methods, we would emphasize that this conclusion is tentative and must be substantiated by further observations.

SUMMARY AND CONCLUSIONS

The energy metabolism of five infants in different states of nutrition was determined in eight comparable observations in the respiration chamber during sleep and during crying. The daily activity of six infants, ranging from 8 to 60 per cent underweight for age, was also recorded accurately for from three to six successive days.

The results afford no evidence that poorly nourished infants are more wasteful of energy than well nourished infants during periods of apparently similar activity. An abnormal energy metabolism accompanying activity, therefore, would not seem to be a consistent or important factor in maintaining the marasmic state.

There is some evidence to indicate that infants in poor nutrition cry and are otherwise active for longer periods of the day than subjects in good nutrition. This state of prolonged activity may render an excessive fraction of the energy of the ingested foodstuffs unavailable for purposes of growth. This may explain the persistently stationary weight of some marasmic infants who are apparently receiving and tolerating an adequate caloric intake without alimentary disturbance or parenteral infection.

The results reported in the last four papers of the present series support the generally accepted view that infantile marasmus is the end-result of chronic inanition induced by repeated alimentary disturbances or by recurrent parenteral infections. No experimental evidence has been presented to suggest that an abnormal energy metabolism plays a usual or important rôle in maintaining the marasmic state.

161 West Sixty-First Street.

OTITIS MEDIA AND MASTOIDITIS IN INFANTS UNDER THREE MONTHS OF AGE*

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For many years, observers have noted that infection localized in some other part of the body is a frequent cause of secondary gastro-intestinal disturbance. Common sites of infection are in the upper respiratory tract, as the nose and throat, the nasal accessory sinuses, the middle ear and the mastoid cells. More recently our attention has been directed to obscure infection in the mastoid antrum as a cause of nutritional and gastro-intestinal disturbances in infancy.

Hartman,[1] in 1898, first suggested a causal relation. In 1904, Preysing [2] stated that severe gastro-intestinal disturbances were due to toxic substances which found their way into the blood stream from ear infections. Since the publication of Maurice Renaud's [3] article on mastoiditis in infants in 1921, many observations have been made, and much has been written on the subject. Numerous observers, such as McDougal, Knauer, Byfield and Floyd, have made important contributions. Marriott,[4] in 1925, stated that the symptoms of hydrolability and gastro-intestinal disturbance are the result of a specific intoxication, also that the symptoms are the result of a streptococcus toxin which seems to have a special affinity for the capillaries of the intestine. Alden and Lyman [5] wrote a comprehensive article in 1926, reporting fifteen cases in which mastoid antrotomy was done by them. Hemolytic streptococcus was the organism found on culture in eleven of these cases.

During the past twenty years, one of us has been following up a large group of babies in the Babies Aid organization and under the care

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1. Hartman: Die Einwirkung der Otitis media der Säuglinge auf den Verdaungsapparat, Ztschr. f. Ohrenh. **34**:1, 1898.

2. Preysing: Otitis media der Säuglinge, Wiesbaden, 1904.

3. Renaud, Maurice: Bull. et mém. Soc. méd. d. hôp. de Paris **44**:1326, 1921.

4. Marriott, McKim: Observations Concerning the Nature of Nutritional Disturbances. Read before American Pediatric Society Washington, D. C., May 5, 1925, Laryngoscope **35**:592-593 (Aug.) 1925.

5. Alden, A. M., and Lyman, H. W.: Mastoid Infection in Infants, Laryngoscope **35**:586-591 (Aug.) 1925.

of the Children's Agency of the Associated Charities of San Francisco. These babies have been seen at regular intervals during health in a well baby nursery and during illness in the Children's Hospital of San Francisco. The mortality rate in this group of babies, during the years from 1922 to 1925, was 6.3 per cent, as compared to 3.5 per cent during the years from 1925 to 1928. During the year 1928-1929, the mortality rate was 1.8 per cent, the lowest on record for this group of babies. Previous to 1925, the most frequent diagnosis made in this group was some form of gastro-intestinal disturbance. The usual symptoms were refusal of food, severe diarrhea, dehydration, vomiting and fever. Some responded to changes in their formulas; others became progressively worse, despite any dietary treatment or supportive measures.

Postmortem examinations were done on nearly all infants who died. Most of the anatomic diagnoses were either infantile atrophy or bronchopneumonia, the diagnosis of the latter being based on microscopic examination. Nothing abnormal was found in the intestinal tract. It had been noted, however, that a large percentage of these patients gave a definite history or had shown signs of a recent upper respiratory infection. Feeling that the true nature of the condition had not been determined, we undertook a more careful study of these cases. Heart blood cultures were taken from every infant immediately after death. These cultures showed the presence of pathogenic organisms in 35 per cent of the thirty-seven cases observed during the years from 1922 to 1925, the most frequent organism isolated being a streptococcus.

Having found a septicemia in a number of cases, we felt that a search must be made for the true source of the infection. In an endeavor to find this focus, we looked to the upper respiratory tract, because of the frequent history or evidence of infection in this region. On careful physical examination, it was found that a large number of these infants, before the onset of the diarrhea, showed a reddened throat and an abnormal appearance of the tympanic membrane, as compared with infants in good health of the same age. More complete postmortem examinations were done, the middle ear and the mastoid being investigated particularly. In a large number of cases, evidence of pathologic conditions in these regions was revealed.

During the past three years, we have studied carefully every case of infantile diarrhea in the light of these observations, and we have found that by early diagnosis and appropriate treatment (tympanic paracentesis, etc.) the diarrhea and general condition in a number of cases rapidly improved. In many of those which did not respond, a diagnosis of mastoiditis was made.

We present a study of twenty-four cases of mastoiditis. In our series the oldest patient was 3 months of age, the youngest 14 days; the

average age being twenty-six days. The average weight was 7 pounds (3.2 Kg.). Of fundamental importance is the fact that practically all of these infants were foundlings, several were premature, and all were artificially fed. Thus we had a group that was probably somewhat below par and therefore more susceptible to infection. Of the twenty-four patients, eight died without operation; on the remaining sixteen, mastoidectomy was done, with eight recoveries. In sixteen of the twenty-four cases we were able to obtain a definite history of an acute upper respiratory infection; in several cases, as long as three weeks before the onset of symptoms of diarrhea and intoxication.

The first symptoms were refusal of food, occasionally vomiting and loose, greenish stools. Nineteen patients showed a definite diarrhea, with from four to twenty-four stools in the twenty-four hours. No macroscopic blood was present, and the stools usually contained a small amount of mucus. As the symptoms progressed, the stools became more frequent and more liquid, with an increase in the amount of mucus. The infant became rapidly dehydrated, and soon presented the picture of marked dehydration; with sunken eyes, rapid pulse, pallor or slight cyanosis, fever and abdominal distention. Usually at the onset of the symptoms, the fever ranged from 99 to 101.6 F. While a high fever is usually seen in this type of case, some decline just as rapidly with little or no fever. Thrush, which did not respond to any method of treatment, was present in four cases, indicating lack of resistance.

On examination of the ears with the electric otoscope, there was found an opaque grayish-looking tympanum in which the light reflex was absent, a reddened drum with or without the light reflex or redness of the tympanum with slight sagging of the superior posterior canal wall. The latter was the most frequent observation. In only one case was there postauricular swelling.

The white blood count varied from 8,000 to 25,000, the average being 16,000, with a neutrophilic polymorphonuclear count of 58 per cent. The average hemoglobin content was 77 per cent. In seven of the cases, the urine showed albumin, occasional hyaline or granular casts and occasional pus cells. The urine was cultured in eleven cases. In five cases cultures showed the same organism later isolated in pus from the mastoid, and in two the same organism that was present in the postmortem heart blood. Pathogenic organisms were found in the heart blood in five of the sixteen fatal cases. Fourteen stool cultures were done. The same organism found in the mastoid was isolated in five cases.

Paracentesis was performed in every case showing any abnormality of the drum membrane. Frank pus was obtained in six cases at the time of paracentesis; of these, five showed the same organism that was

later isolated from the mastoid. Bilateral involvement of the mastoid was found either at operation or at the postmortem examination in seventeen of the twenty-four cases and unilateral involvement in seven cases. In many of the cultures from pus found in the mastoid more than one organism was found. The organisms in the order of their frequency were hemolytic streptococcus, nonhemolytic streptococcus, *Streptococcus viridans*, pneumococcus, staphylococcus and *Bacillus coli*.

TABLE 1.—*Results of Cultures in Fatal Cases with Mastoidectomy*

No.	Ear Culture	Mastoid Culture	Postmortem Culture
45708	Right ear, Staphylococcus aureus, streptococcus; left ear, Staphylococcus aureus, streptococcus	Right ear, negative; left ear, hemolytic streptococcus, Staphylococcus aureus, a small gram-negative rod	Right ear, hemolytic streptococcus, B. coli; left ear, hemolytic streptococcus, B. coli
46442	Right ear, nonhemolytic streptococcus; left ear, nonhemolytic streptococcus, Staphylococcus albus	Right ear, few staphylococci; left ear, few staphylococci	Right ear, nonhemolytic streptococcus, B. coli; left ear, nonhemolytic streptococcus, B. coli
45429	.....	Right ear, negative; left ear, Staphylococcus aureus	Right ear, hemolytic streptococcus; left ear, hemolytic streptococcus
45975	Right ear, hemolytic streptococcus, gram-positive rod; left ear, hemolytic streptococcus, gram-positive rod	Right ear, hemolytic streptococcus, Staphylococcus aureus; left ear, hemolytic streptococcus and B. coli	Right ear, hemolytic streptococcus, staphylococcus; left ear, hemolytic streptococcus, staphylococcus
48190	.....	Right ear, negative; left ear, Streptococcus viridans, B. coli	Right ear, Streptococcus viridans; left ear, Streptococcus viridans, B. coli
46519	Right ear, hemolytic streptococcus, Staphylococcus albus; left ear, Staphylococcus albus	Right ear, hemolytic streptococcus; left ear, B. coli	Right ear, hemolytic streptococcus and B. coli; left ear, B. coli
49312	Right ear, Staphylococcus albus; left ear, gram-positive sporulating rod	Right ear, Staphylococcus albus and B. coli; left ear, B. coli and gram-positive rod	
49003	.....	Right ear, negative; left ear, negative	Right ear, Streptococcus viridans, Staphylococcus albus, B. coli; left ear, Streptococcus viridans, Staphylococcus albus, B. coli

Roentgenograms of the mastoid region were taken in several cases, but no evidence of a pathologic process was present. Roentgenograms of the chest were taken in thirteen cases. In only three was any abnormality seen, two showing a beginning bronchopneumonia and one enlarged bronchial glands.

Mastoidectomy was done in sixteen cases. These were done by various members of the ear, nose and throat staff of the Children's Hospital. All of the operations were performed under local anesthesia, and none of the deaths could be attributed to the operation itself or to postoperative shock, in spite of the fact that a number of babies were in extremis when operated on. Definite involvement of the mastoid

TABLE 2.—*Fatal Cases with Mastoidectomy*

No.	Age	Past History Upper Respiratory Infections	Observations on Ear	Clinical Course	Operation	Anatomic Diagnosis
45975	6 weeks	Rhinitis and conjunctivitis 2 weeks before entry to hospital	Both posterior canal walls reddened	Diarrhea, loss of weight, refusal of food; moribund on entry to hospital	Bilateral paracentesis and bilateral mastoidectomy; pus in both middle ears and both mastoids	Bilateral mastoiditis and otitis media; bronchopneumonia; acute splenic tumor; hyperplasia mesenteric and retroperitoneal lymph glands
49343	7 weeks	Not known	Right ear, posterior canal wall reddened, tympanum reddened and bulging; left ear, typanum reddened	Diarrhea, vomiting, fever, 99 to 103 F., nasal discharge	Bilateral paracentesis; right middle ear contained pus; left ear contained serosanguineous material; bilateral mastoidectomy; both mastoids contained mucorpurulent material	Bilateral mastoiditis and otitis media; thrombosis of both transverse sinuses
49003	13 days	Not known	Right ear, redness of tympanum and posterior canal wall; light reflex absent; left ear, redness of tympanum and canal wall with sagging of superior posterior portion	Severe diarrhea; refusal of food; fever 100.2 to 101 F.	Bilateral mastoidectomy; no pus obtained.	Bilateral otitis media and mastoiditis; emphysema and atelectasis; recent operation for mastoid drainage; one-half of mastoid cells on both sides are removed
46442	2 months	Not known	Right ear, slight dulling of tympanum; light reflex present; left ear, slight reddening of tympanum and posterior canal wall; light reflex present	Diarrhea, vomiting, temperature ranged 99.6 to 107 F.	Bilateral paracentesis; bilateral mastoidectomy, bloody serum obtained	Bilateral otitis media and mastoiditis. Description: A simple mastoid operation was performed; the bone just anterior to the mastoid cells was removed; on the right the middle ear and mastoid contained serous and hemorrhagic exudate; operation on the left side opened the anterior one-third of the mastoid cells, which contained hemorrhagic exudate; the bone was soft and friable
46549	2 months	Patient had red throat two weeks before onset of symptoms	Right ear, tympanic membrane reddened with slight bulging of posterior canal wall; left ear, negative	Loss of weight, diarrhea, fever and vomiting	Bilateral paracentesis; right ear, frank pus obtained; left ear, serosanguineous material obtained; bilateral mastoidectomy; right ear, pus present; left ear, negative	Bilateral otitis media and mastoiditis. Description: A simple mastoidectomy was done on both sides; about one-half of the mastoid cells were removed; the remaining cells were soft and hemmorrhagic
45708	3 weeks	Not known	Right ear, negative; left ear, dulness of tympanum, no light reflex present	Diarrhea, vomiting, fever 100-106 F.	Bilateral paracentesis; bilaterial mastoidectomy; bloody serum obtained at both operations	Bilateral otitis media and mastoiditis; edema and congestion of lungs, marked atrophy of thymus
45429	1 month	Not known	Right ear, tympanum slightly reddened; left ear, negative	Diarrhea, loss of weight	Bilateral paracentesis; right ear, serosanguineous material; left ear, negative; bilateral mastoidectomy; no pus found	Bilateral suppurative otitis media and mastoiditis; bilateral bronchopneumonia
48321	3 months	Discharged from hospital 12 days before onset of present illness; diagnosis: subacute tonsillitis, rickets and secondary anemia	Negative	Diarrhea 5 days; fever 99-106 F.	Bilateral paracentesis and mastoidectomy; no pus found	Bilateral mastoiditis; rickets

TABLE 3.—*Fatal Cases Without Mastoidectomy*

No.	Age	Past History	Observations on Ear	Clinical Course	Operation	Anatomic Diagnosis
46934	19 days	"Cold" and red throat 9 days before onset of illness	Bilateral paracentesis before entrance to hospital; right ear, dried blood in canal; left ear, discharging pus	6-8 stools daily; temperature 101.8 F. on entry, with terminal rise to 105 F. 2 days later	Bilateral paracentesis; right ear, dried blood in canal; left ear, pus obtained	Bilateral acute suppurative otitis media and mastoiditis, confluent bronchopneumonia, acute splenic tumor
48610	1 month	Reddened throat and fever on admittance to baby nursery, 3 days previous to entry	Right ear, redness of posterior canal wall with dulling of tympanum; left ear, reddened tympanum and canal wall	Loss of weight, vomiting and fever, diarrhea	Bilateral paracentesis; right ear, serosanguineous material; left ear, serosanguineous material	Bilateral otitis media and mastoiditis; diffuse bronchopneumonia
53171	6 weeks (Premature)	Dismissed as well from hospital at age of 3 weeks, gaining well; returned four days later with loss of weight	3 weeks after second entry to the hospital both canal walls and tympanic membranes congested, no bulging	10 days after entry to hospital developed a vaginal discharge; smears showed B. coli and streptococcus; at the same time developed severe diarrhea, temperature ranged from 99.6 to 100.6 F.	None	Bilateral otitis media and mastoiditis; congestion both bases with early signs of bronchopneumonia; abscess right kidney, acute vulvovaginitis, acute pericarditis.
47771	3 months	In Children's Hospital for 3 days' observation before entry to well baby nursery; diagnosis, subacute tonsillitis	Opaque "ground glass" appearance both drums	3 days after dismissal from hospital reentrance in a severe convulsion with fever 105.6 F.; expired 3 hours later	None	Bilateral otitis media and mastoiditis; pulmonary congestion and edema of left lower lobe
49819	17 days	Jaundice since 3 days of age; had never taken food well	Right ear, redness and slight bulging of tympanum; left ear, redness of posterior canal wall, tympanum reddened and bulging	Diarrhea, vomiting, temperature range, 99 to 106 F.	Bilateral paracentesis; right ear, serosanguineous material; left ear, same	Bilateral otitis media and mastoiditis; both middle ears and mastoids filled with pus
49220	21 days	Not known	Both tympanic membranes red and bulging; posterior canal walls red and bulging	Diarrhea, fever and listlessness	Bilateral paracentesis; right ear, pus obtained; left ear, same	Postmortem operation showed much thick pus in both mastoids
48096	5 weeks	Had acute tonsillitis and thrush two weeks before onset of illness	Right ear, slight dulling of tympanum, no light reflex present; left ear, redness of tympanum and posterior canal wall	Diarrhea (severe), marked dehydration, fever 99.6 to 104 F.	Bilateral paracentesis; serosanguineous material obtained from both	Otitis media and mastoiditis (right ear); diffuse bronchopneumonia, purulent bronchitis
49465	1 month	Not known	Both tympanic membranes reddened, with bulging of both posterior canal walls; light reflex absent	Liquid stools 3 days; fever 101 F.; cyanotic and markedly dehydrated	Bilateral paracentesis; right ear, serosanguineous material; left ear, same	Bilateral otitis media and mastoiditis; extensive thrombosis of right lateral sinus; bronchopneumonia of left lower lobe

cells was reported in most instances. In the fatal cases in which no involvement of the mastoid had been found at operation, it was discovered at postmortem examination that the mastoid cells had either been aberrant and had not been entered, or had been only partly removed. Those which remained were found to contain pus and granulation tissue.

In most cases we were able to do our postmortem examination shortly after death. The approach to the mastoid region was made from within by way of the posterior fossa. The cortex of the petromastoid portion of the temporal bone was carefully removed, thus exposing the mastoid cells. Contrary to the general opinion, in the majority of cases, several cells were present. In no case was any abnormality found in the gastro-intestinal tract. Involvement of the lungs ranging from slight congestion of the bases to definite bronchopneumonia was found in eight of the sixteen cases that came to autopsy.

In all cases, special attention was paid during the preoperative and the postoperative course to the maintenance of the body fluids by hypodermoclysis and by forcing fluids by mouth. Some were fed on a high carbohydrate prepared food which was bland and easily assimilated; others were fed various protein milk mixtures. There was no appreciable difference in the number of stools or the clinical course in the two groups, nor did a change from one type of food to the other alter the course until after the source of the infection had been removed. Small transfusions were given in a number of cases following operation, which proved to be a valuable part of the general supportive treatment.

COMMENT

Although this paper deals with a group of infants who were physically below par before the onset of their illness, and therefore had little power to resist infections, we cannot overlook the fact that sometimes the more favorably situated infant seen in private practice suffers in the same way. It is not uncommonly observed that normal infants, including those who are breast fed, are subject to severe toxic diarrheas which have their origin in the upper respiratory tract. Careful examination reveals reddened throats and altered tympanic membranes. Occasionally, the infection may be so severe that it progresses to the stage of purulent otitis media. In such instances, relief comes only after paracentesis. It is unusual, in our experience, to find the infection extending to the mastoid region in the latter type of infant.

The diagnosis of otitis media and of mastoiditis in the young infant should be made by the pediatrician who is making constant observations and who is guided as much by the clinical picture as by the local conditions in the ear. The otologist rarely has had sufficient occasion to

TABLE 4.—*Cases Recovered with Mastoidectomy*

No.	Age	Past History	Observations on Ear	Clinical Course	Operation	Culture	Clinical Diagnosis
38316	3 months	Birth weight 6 lbs.; entry weight 9 lbs.; breast fed 1 month, vomiting since 1 month of age; has had four colds since birth	Ears negative on entry to hospital; 12 days after entry, right ear, tympanic membrane reddened; left ear, tympanic membrane reddened and bulging; paracentesis left ear, pus obtained	Temperature on entrance 98.8 F.; vomited frequently after feedings; two weeks after entry temperature 103.8 F.; physical examination at that time showed reddened nasopharynx and tympanic membranes; same time patient had 6-7 loose stools daily; paracentesis left ear, pus obtained, fever high for several days following paracentesis and mastoidectomy, then normal	Left mastoidectomy; pus present in mastoid cells	Culture left mastoid, Streptococcus viridans	Malnutrition; rickets; acute nasopharyngitis; bilateral otitis media; left mastoiditis
40092	6 weeks	Birth weight 8 lbs., 8 oz., entry weight 8 lbs., 11 oz.; vomiting and diarrhea 4 days	On entry ears negative; two weeks after entry, both tympanic membranes reddened; paracentesis of left ear, pus obtained	Temperature ranged 99.6 to 101 F. and patient continued to vomit; two weeks after entry, severe diarrhea; at that time, both ear drums reddened; paracentesis of right ear, pus obtained; fever gradually subsided and stools improved after mastoidectomy	Bilateral mastoidectomy; right ear, pus present; cells spongy; left ear, large amount of pus present	Right ear, no growth; left ear, pneumococcus	Bilateral otitis media and mastoiditis
49332	1 month	"Cold" 2 weeks before entry	Right ear, tympanic membrane red and bulging; left ear, tympanic membrane reddened; paracentesis; serosanguineous material obtained	Temperature ranged 99.2 to 104.6 F.; bilateral mastoidectomy performed the day after entry; at the same time, a blood transfusion given; fever subsided and stools improved rapidly	Bilateral mastoidectomy; pus in right mastoid; left mastoid, negative	Right mastoid, Staphylococcus albus; left, negative	Bilateral otitis media; mastoiditis, mastoiditis right
46975	5 weeks	Refusal of food one week; diarrhea 3 days	Right ear, tympanic membrane "ground glass" appearance with redness of posterior canal wall; left ear, tympanic membrane "ground glass" appearance; posterior canal wall slightly reddened and bulging; bilateral paracentesis; pus obtained from left ear	Temperature on admittance 98.6 F., next day, 101 F.; same day bilateral mastoidectomy done; after operation temperature ranged 101 to 104 F. for 10 days; stools improved in consistency 48 hours after operation, with a decrease in number	Bilateral mastoidectomy; right ear, granulation tissue and spongy bone; left ear, pus in mastoid cells	Right mastoid, hemolytic Staphylococcus aureus; left mastoid, hemolytic Staphylococcus aureus	Bilateral mastoiditis; purulent otitis media, right; left ear, serous otitis media; subacute nasopharyngitis

48953	2 months	"Cold" 3 days; diarrhea	Right ear, tympanic membrane reddened; light reflex absent; left ear, tympanic membrane reddened; redness of posterior canal wall; paracentesis, left ear, tympanum pus obtained	Paracentesis left ear, tympanic membrane, day after entry; mastoidectomy same day, stools improved rapidly the 3rd postoperative day; patient had only one stool, which was of good consistency	Bilateral mastoidectomy; pus present in left mastoid	Left mastoid, Staphylococcus aureus and Staphylococcus albus	Bilateral otitis media; mastoiditis, left
50108	1 month	Diarrhea 3 days; vomiting 2 days	Right ear, tympanic membrane red and bulging; redness and bulging posterior canal wall; left ear, same	Temperature ranged 99.6 to 104 F.; 6 to 7 liquid stools daily; bilateral mastoidectomy 5 days after entry; temperature normal 24 hours after operation and remained normal; vomiting ceased and stools improved rapidly	Bilateral mastoidectomy; pus in right mastoid; left mastoid negative	Culture right ear, B. coli; left ear, no growth	Acute nasopharyngitis; bilateral otitis media; mastoiditis, right
41445	4 weeks	Birth weight 10 lbs.; present weight 8 lbs., 12 oz.; breast fed, vomiting since birth, projectile at times; failure to gain weight	4 days after entry, physical examination revealed reddened nasopharynx and reddened ear drums; 5 days later, paracentesis of right ear, tympanic membrane showed no discharge	Temperature 99 F. on entrance; 4 days later, 104 F.; physical examination showed red nasopharynx and reddened ear drums; vomiting continued, projectile at times	Right mastoidectomy 11 days after entry; pus present in right mastoid; Fredet Rammstedt operation 17 days after entry; pyloric tumor found	Right mastoid, pneumococcus; left mastoid, negative	Bilateral otitis media; right ear, mastoiditis; pyloric stenosis
32115	3 months	Pertussis for 6 weeks, loss of weight, vomiting	Right ear, tympanic membrane dull and reddened; left ear, tympanic membrane dull and reddened; bilateral paracentesis; serosanguineous material obtained	Temperature on entrance 100 F.; third day after entrance, 104 F.; bilateral mastoidectomy and transfusion the same day; slow improvement, fever subsided; 10 days after mastoidectomy, diffuse, bronchopneumonia, following this, gradual improvement	Bilateral mastoidectomy; pus found in left mastoid; right mastoid, negative	Left ear, streptococcus viridans; nonhemolytic streptococcus	Left ear, mastoiditis and otitis media; diffuse bronchopneumonia

examine many very young infants, and therefore has not had sufficient opportunities to make him familiar with the evidence of otitis media and mastoiditis which appears valid to the pediatrician in the case of the very young infant.

At the present time, careful otologic observations are being made independently by two competent otologists on this group of foundlings during health as well as during illness, in the hope of establishing definite local signs whereby an early diagnosis of involvement of the mastoid can be made in the young infant. We believe that early paracentesis of the tympanic membrane is an important factor in relieving symptoms, and we have found that pus is often obtained when little evidence of its presence is found on examination of the drum. The finding of no pus, however, does not preclude the presence of involvement of the mastoid, as shown in our cases.

Our experience in handling a large group of infants has been similar to that of pediatricians elsewhere. A number of years ago, we made the diagnosis of gastro-enteritis in our cases of severe diarrhea much more frequently than we do at the present. The evolution of these observations has been divided into three periods. It was during the first period that we began to feel that the true nature of the condition was being overlooked. The results of culture of the heart blood showed that septicemia was frequently present in a large number of cases. More complete postmortem examination revealed the middle ear and mastoid as the apparent foci of the infection. More careful clinical study brought out the fact that infections of the upper respiratory tract frequently preceded or accompanied the condition.

With these discoveries, we enlisted the cooperation of the otolaryngologist and entered the second phase of our study. In the beginning, because of obscure local signs in the ear, we found them skeptical and reluctant to operate in cases in which we had made a diagnosis of mastoiditis and otitis media. However, the results of early paracentesis and mastoidectomy thoroughly confirmed the lessons learned at postmortem examinations, and by this treatment a large number of intoxications of early infancy were prevented.

The third period is marked by increasing interest and cooperation on the part of those interested in otology, with the result that by early diagnosis, early paracentesis and mastoidectomy, if necessary, the proportion of cases showing this intoxication syndrome has greatly decreased, and the diagnosis of primary gastro-enteritis is seldom made. This, to us, seems to be an important factor in the low mortality rate in this group of infants during the past year.

CONCLUSION

Many diarrheas of infancy are toxic and originate from infections of the upper respiratory tract, particularly in the middle ear and mastoid.

In all cases of infantile diarrhea which do not readily respond to dietary management, careful examination of the middle ear should be made. Early paracentesis of the tympanic membrane is indicated whenever any evidence of infection of the middle ear exists. If rapid improvement does not follow paracentesis and supportive measures, involvement of the mastoid is almost certain. Early operation with care taken to find and to open all of the mastoid cells involved offers the best chance for recovery and involves but little risk.

MALNUTRITION

A STUDY OF PREVENTORIUM TREATMENT AND OF END-RESULTS*

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Camp Hillcrest at Fayetteville, 8 miles from Syracuse, N. Y., was established in 1919 as a summer camp for undernourished children. By 1922, it had developed into a preventorium accommodating forty children, and remaining open all the year. Two years later, a wing was added with fifteen beds for crippled children, chiefly for those suffering from the after-effects of poliomyelitis. Camp Hillcrest is now a well equipped institution with 6 acres of high ground, vegetable and flower gardens, a shallow swimming pool and adequate space for play and recreational activities. The children sleep in open-window pavilions and attend school in open-window classrooms. The camp has a capable resident nursing and operating personnel, the medical and dental care being furnished by a staff of physicians and dentists from Syracuse and Fayetteville.

Girls from 6 to 14 and boys from 6 to 12 years of age are admitted for the treatment of malnutrition. Occasionally, patients with cardiac disorders, convalescents or patients with miscellaneous conditions are accepted. The orthopedic cases are sent to the wing for crippled children, where the patients receive muscle training in a warm water bath, and other special treatment. Applications for the admission of undernourished children came mostly through school nurses or physicians and occasionally through other agencies or from private physicians. The home conditions are investigated by a nurse from the bureau of nursing of the health department, the social and medical history forms are filled out, and the child is then taken to one of the examining physicians of Hillcrest, usually to the chest clinic at the Syracuse Free Dispensary. A careful physical examination is made with particular reference to the nutritional status, and a routine intradermal tuberculin test is performed. If a positive reaction is obtained or the physician otherwise desires it, a roentgenogram of the chest is made. No patient with active tuberculosis is accepted.

It has always been the aim of the preventorium to have physical defects corrected as fully as possible before admission so that the children may be free to gain, without obvious physical handicaps. From

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* Retiring President's Address, Syracuse Academy of Medicine, Jan. 29, 1929.

the practical standpoint so many difficulties are often encountered, that part or sometimes all of the correctional work has to be done after admission. Dental care and the removal of diseased tonsils and adenoids have constituted the major part of the correctional work. The wholesale removal of tonsils has never been approved at Hillcrest, but where there is fair reason for believing that the condition of the tonsils or adenoids is definitely interfering with the child's well-being, the staff has felt justified in recommending surgical measures. On admission, a culture of the throat, urinalysis and a vaginal smear are required. A routine Wassermann test reveals an occasional case of syphilis. If there are no active lesions, the children are accepted if in need of care at Hillcrest, but are taken to the Syracuse Free Dispensary for treatment. To attempt to build up a child's nutrition without trying to find out if there might be an underlying syphilis, would obviously be unsound practice.

Most of the children begin to put on weight soon after admission, and some truly amazing gains have been recorded. A few children fail to gain normally in spite of a nutritious diet and attention to various important details. Even an intensive study of some of these children fails to explain their lack of normal progress. Many of them are of a neurotic type and do not eat or rest as well as the other children. Some probably have latent forms of tuberculosis which have eluded search. In other cases, time alone can bring about a normal state of nutrition, and in a few children this goal is never attained.

A majority of the children at Hillcrest reach a normal state of nutrition in approximately six months. A few remain a year or even longer. After they have reached the point when they no longer need care at Hillcrest and are ready to return home, a large portion of the problem still lies ahead. Will these children retain the gain they have made and continue to practice the health-giving habits of living and eating which they have learned, or will they succumb to an unfavorable environment, poor food and faulty habits? This problem was recognized early in the work, and four years ago sufficient funds became available to employ a full time worker, one of whose duties was to take charge of the after-care of Hillcrest graduates. Visits to the homes were made, classes for both mothers and children were held, and the weights of the children were recorded. For one worker, however, the task was too great, and on July 15, 1928, the generalized nursing service of the Syracuse Health Department took over this phase of the work at Hillcrest. The nurses not only make the social investigations leading up to the admission of children to Hillcrest, but also have charge of the follow-up work on these same children after discharge. It

is too early to make an appraisal of the value of the new type of work, but it is hoped that the results will prove more satisfactory.

During the school year a short school session is held twice a day. Following the morning class, a short rest period is allowed before dinner and a longer one of an hour and a half after dinner, when most of the children go to sleep. During the vacation period in the summer, they are outdoors the greater part of the time and play regulated games, go splashing in the wading pool, listen to storytelling and fill their days very full. Much rest and a limitation of activity are insisted on as essential for malnourished children. The homelike atmosphere pervading Hillcrest destroys the impression of an institution. The children are a happy group who thoroughly enjoy their progress on the road to health. In the summer all but the oldest children wear only bathing trunks, and an effort is made to realize the most possible from exposure of the body surface to sunlight. In the darker months of the year, treatments with the Alpine lamp are given to those most in need of them.

Usually undernourished children have faulty posture. This is felt to be a matter of considerable importance, and attempts have been made at correction by postural exercises given each school morning by the two school teachers, according to the Goldthwait method. Care is taken to see that the exercises are not carried to the point of fatigue. Whenever possible during the day, the children are encouraged to assume the correct posture, standing or sitting.

The Baldwin-Wood tables of height and weight, published by the American Child Health Association, are used in estimating the percentage of underweight, if present. A great deal of discussion has taken place as to the value of height and weight tables in estimating the nutritional status of children.

Emerson [1] is perhaps one of the most outspoken advocates of the value of these tables and goes so far as to state that any child who is habitually 7 per cent underweight for a given height will show evidence of malnutrition. In reply to a personal communication, Dr. C. E. Hayes of the American Child Health Association wrote that the Association sponsors the weight-height tables as the best extant. She called attention, however, to the recent work of Franzen,[2] based on numerous measurements of children 11 and 12 years of age. His studies indicate that a child who appears underweight in the usual tables may not be unusual for children having a similar bony framework. He expressed the belief that much of the departure from average weight for height is due to difference in skeletal width or depth. A method is being

1. Emerson, P. W., and Manny, F. A.: New Weight-Height Tables and Malnutrition, Arch. Pediat. **41**:677 (Oct.) 1924.

2. Franzen, R.: Child Health Bull. **4**:176 (Nov.) 1928.

worked out depending on four variables, the breadth and the width of the chest, the width of hips and the height. He stated that deviations from predicted weight are not necessarily in themselves diagnostic of health, and stressed the importance of clinical opinion.

Clark, Sydenstricker and Collins [3] reported data on 506 children who, on detailed examination by medical men experienced in this type of work, were found to be without evidence of disease and in a good or excellent state of nutrition. According to the Baldwin-Wood tables, 16 per cent of these healthy children were found to be 10 per cent or more underweight. These authors felt that their results throw doubt on the value of the tables in assessing physical fitness and they believed that the method should be examined further. In another paper,[4] they gave details of an extensive experiment involving the examination of nearly 10,000 school children from 6 to 16 years of age. Their status of nutrition was classified according to clinical evidence as excellent, good, fair or poor. Forty per cent of the "poor" nutrition group fell within the assigned normal limit of 10 per cent variation and would have been entirely missed if the tables alone had been considered.

From a study made in an Italian quarter of New York City, Dublin and Gebhart [5] concluded that the height-weight tables were of little or no value in the selection of undernourished Italian children. By the use of the standard tables, three quarters of the children were overlooked whom the physician, after a thorough examination, pronounced undernourished. The authors expressed the belief that the height-weight tables are useful in selecting a considerable group for study, but that a diagnosis of malnutrition should be made only after a careful examination by a competent physician.

Davenport [6] studied body build and its development from the standpoint of inheritance, and noted that a tendency to slenderness or fleshiness runs in families, sometimes only in certain members. There is an internal biochemical difference which affects metabolism. Some persons gain largely in fat and become obese, others metabolize food more largely into protein and remain slender. That there is a difference in metabolism is shown by the difficulty in reducing the weight of certain persons even when placed on a diet low in calories. Constitutional as

3. Clark, T.; Sydenstricker, E., and Collins, S. D.: New Baldwin-Wood Weight-Height-Age Tables as an Index of Nutrition, Pub. Health Rep. **39**:518 (March 14) 1924.

4. Clark, T.; Sydenstricker, E., and Collins, S. D.: Weight and Height as an Index of Nutrition, Pub. Health Rep. **38**:39 (Jan. 12) 1923.

5. Dublin, L. I., and Gebhart, J. C.: Monograph, N. Y. Association for Improvement of Conditions of the Poor, 1924.

6. Davenport, C. B.: Body Build and Its Development and Inheritance, Carneg. Inst., Washington, Bull. no. 24.

well as nutritional differences determine body build. Geographical and racial differences are important. Davenport's conclusions are based on extensive family studies of the inheritance of slenderness and fleshiness. He also stated that variations in the chromosomes, in accordance with mendelian laws, lead to slenderness or fleshiness in the progeny.

The combined method of appraising the home surroundings and history, making a careful medical examination and estimating the height-weight index is used at Hillcrest. The physician's examination is made with particular reference to such conditions as apathy, pallor, circles under the eyes, flabby skin or muscles, lack of subcutaneous fat and faulty posture. To depend on weight-height tables alone would often lead one astray, but when the tables are taken in conjunction with other factors, they have proved of considerable value. The tables possess two definite merits; they are simple, and they eliminate the personal equation. It is generally conceded that they are far from ideal, but in the future they will undoubtedly be amplified and improved.

The tables are of even greater value in estimating the nutritional progress of the individual child. Instead of the usual temperature chart, a weight chart for each child hangs on the wall of the physician's examining room at Camp Hillcrest. A point is found at the left of the chart indicating the proper weight for height and age, from which a straight red line is drawn, sloping gradually upward to indicate a gain of one-half pound (0.25 Kg.) a month. The child's actual weight each week is plotted in a black irregular line, usually considerably below the hypothetical red line of normal gain in weight. When the black line of actual weight gradually ascends and finally reaches the red line, the child is considered to have obtained the theoretical normal weight for its age and height. The children thus have a visible demonstration of their progress and are encouraged to further their efforts. The method is crude, as one-half pound per month is arbitrarily taken as the proper gain regardless of age or sex, and the Wood tables are used as a definite standard for optimum weight. Nevertheless, the procedure has been found to be of considerable practical advantage. Every six months the height is again taken, and a new red line is plotted.

A study was made of a group of 100 children who had remained at Camp Hillcrest from three months to one year, and had been followed, after leaving camp, for from one to three years. Children were excluded who had stayed at Hillcrest only a few weeks, or who had not been under their usual home influence for at least a year after graduation. This special group was studied in the home by a trained nurse who had just resigned as superintendent of Hillcrest after four years of service, and who knew all the children intimately. The physical examinations were made by three physicians of Hillcrest who are

especially trained for this work. As chief of the medical staff, I directed the experiment.

Chart 1 shows that 54 of the 100 children fell within the 10 per cent variation allowed for the theoretical normal weight for height, but for one reason or another were considered in need of care at Hillcrest. Ninety-two children left camp with normal weight, but from one to three years later this number had dropped to sixty-seven. Conversely, the right half of the chart shows that only eight children went home 10 per cent or more underweight, and that when a check was made later, thirty-three were found to be underweight. In view of the splendid regimen under which these children lived at camp, and the amount of

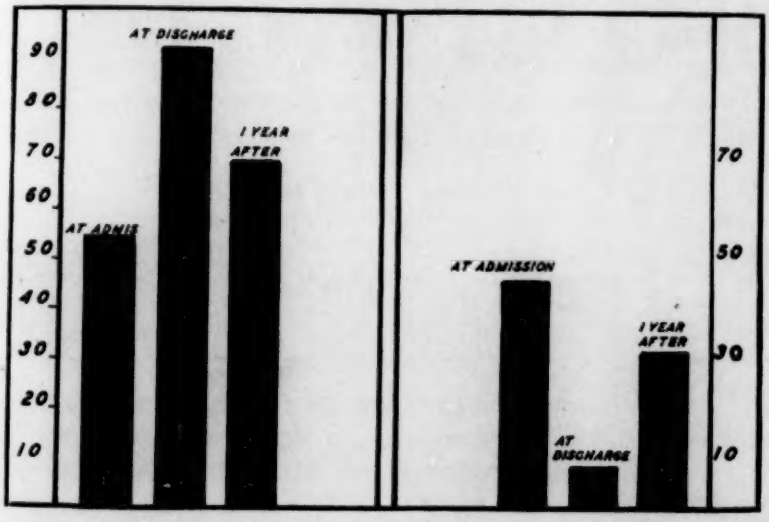

Chart 1.—Statistics for normal and underweight children at Camp Hillcrest. The left column indicates condition of normal children; the right column, those 10 per cent or more underweight.

health education they received, these results were a bit disappointing. They emphasize the need for a more intensive supervision of the children and their home environment after graduation.

The table gives a comparison of the nutritional status of the children, as determined by the examining physician, with their weight-height index, both on admission and during the follow-up study. Where the nutritional status reported by the examining physician is good, the agreement with the weight percentage is very close. More discrepancy is seen in the passable group, while in those children listed as poor, eighteen of the group on admission, and twelve children one year or more later, would have been considered within normal limits if only the

weight tables had been consulted, and no physical examination had been made.

Chart 2 represents a study of the health habits of eighty-four of the children. An improvement is shown in most of the important items listed save in two noteworthy instances, rest during day, and rest at night. The lack of sufficient rest at home was undoubtedly a prominent factor in sending down grade some of the children who had left Hillcrest in excellent condition.

Chart 3 is the weight chart of Evelyn P. as it hung on the wall at Camp Hillcrest. Her mother died of tuberculosis a few years before, and the child was being cared for by a housekeeper employed by her father. The patient entered Hillcrest in June, 1926, aged 10, 17 per cent underweight. She gained weight in an almost spectacular fashion, 23 pounds (10.4 Kg.) during her year's stay, and was discharged in fine health with weight a little above the theoretical normal. The follow-up worker kept in touch with Evelyn, but when a year and four months later she was reexamined, she was found to be 13 per cent under-

Nutrition Status Compared with Weight

Nutritional Status	Weight on Admission		Weight 1 Year After	
	Normal	10% Under	Normal	10% Under
Good	11	1	28	0
Passable	25	5	23	4
Poor	18	30	12	28

weight. This case is quoted to show the form of weight chart used, and to emphasize the prime necessity for vigilant after-care if the good results of preventorium treatment are to be maintained. However, even though a good state of nutrition may to a certain extent be lost in some children, many intangible benefits that cannot be weighed or measured must remain. These children in their most impressionable years, undoubtedly carry home with them enduring memories of the wonderful atmosphere at Hillcrest, with its cleanliness, right living, good health habits and proper training of mind and body. Some of them return home as little missionaries of good health.

Bush and Shepherd [7] reported less favorable results from Del Valle Preventorium, Alameda County, Calif. Of 107 children followed for two months to two years after leaving the preventorium, 54 per cent were found to be underweight. The authors felt that the striking thing needed by these children was additional rest, which they usually failed to get because of the ignorance or poverty of the parents or because of the lack of parental control. To overcome this defect in the after-care of preventorium children and also to care for undernourished chil-

7. Bush, C., and Shepherd, W. P.: Nat. Tuberculosis Assoc., June, 1928.

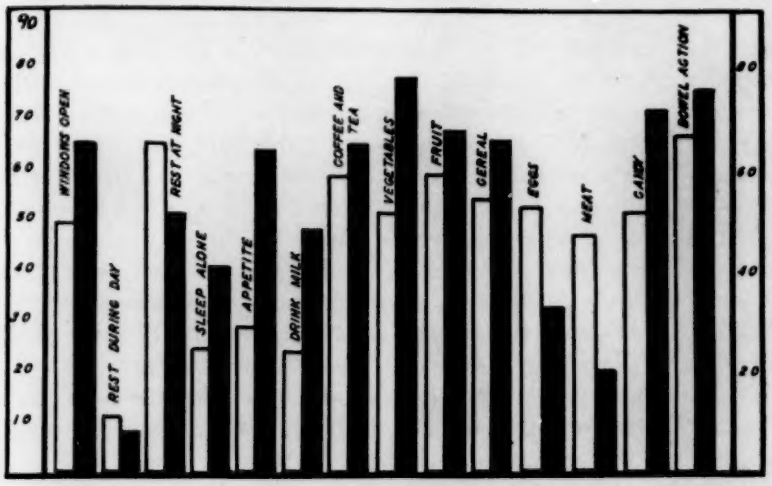

Chart 2.—Summary of health habits of eighty-four children on admission and one year or more after leaving Camp Hillcrest. The white column indicates a summary of the habits of children on admission; the black column, habits one year after leaving.

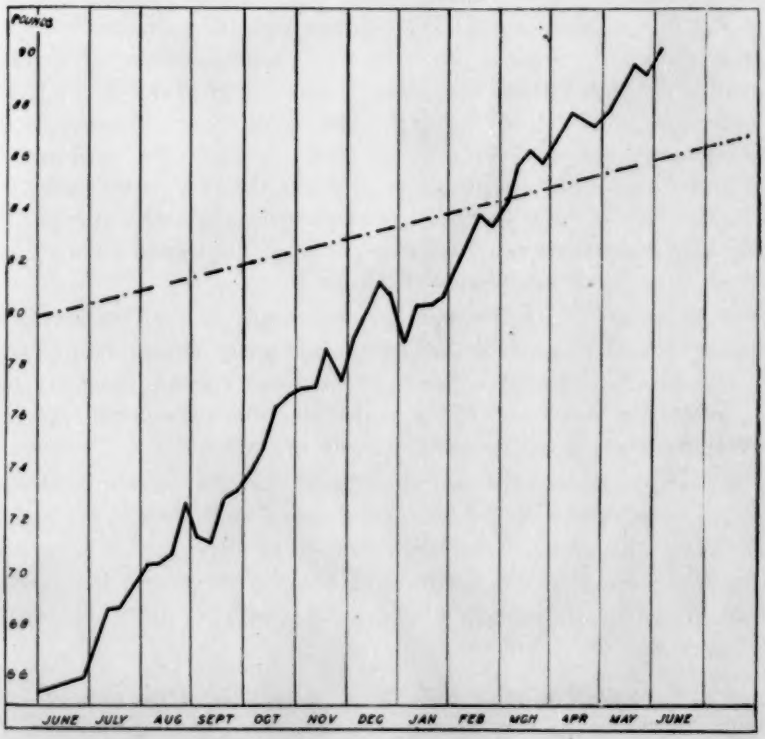

Chart 3.—Weight chart of E. Petrie; aged 12, height, 56½ inches, and weight, 66 pounds.

dren and convalescents from acute illnesses, a sunshine school was started in Berkeley, Calif., by Dr. W. P. Shepherd. Using open-air classes as a type, he amplified their regimen by adding an hour's daily exposure to sun or air together with two additional hours of rest and an intensified health program. Many children discharged from the preventorium were sent to the sunshine school for a time before returning to the ordinary classrooms. It was found that 70 per cent of the children previously listed for admission to the preventorium were brought to good health standards by the sunshine school. This was accomplished at an additional cost over regular school expenses of 17 cents per day per child as compared with a cost of $1.20 per day per child at the preventorium. The authors expressed the belief that the preventorium should concentrate only on those children not benefited by open-air schools, and that emphasis on rest rather than fresh air, food or sun is the keynote of success.

In Boston, the regulation open-air classrooms have been found to be an unsatisfactory solution of the problem, and a series of nutrition classes for physically substandard children requiring rest and supplementary nutrition have recently been instituted. A room is set aside in each of fifteen schools where children from various classes are brought together for a midmorning rest on cots and for a lunch, the recess lasting forty-five minutes. At noon they again assemble for a rest of fifty-five minutes, followed by dinner. Each child pays for the food at an average cost of 11 cents a day. The children are not segregated in special rooms but carry on their scholastic work in the various classrooms with very little interference, because the rest periods are timed to replace the morning and noon recesses and gymnasium periods. No special open-air teachers are required, and any number of children from various grades can be accommodated.

In Montclair, N. J., "comparison was made of the outcome of the open-air school and of an ordinary classroom, where children were given a specially adapted hygiene program with rest and noonday feeding, as carried out in the outdoor school. No perceptible advantage was apparent from either method over the other." [8]

Reports from various sources indicate that the conventional open-air classroom, where the children wear special clothing and are exposed to outdoor atmosphere, is tending to disappear. Its place is being taken by more simple rest and nutrition classes, or by general improvement of the hygiene of the school day, particularly with regard to rest, feeding and open-window ventilation.

8. Personal communication from Dr. L. A. Wilkes of the American Child Health Association.

CONCLUSIONS

1. Mild forms of malnutrition can be satisfactorily treated by a proper regimen in the public schools.

2. Preventorium care with its greater cost should be reserved for the more severe cases.

3. Intensive follow-up work is necessary for permanent results, whatever type of treatment is chosen.

MINIMUM REQUIREMENT OF CALCIUM AND PHOSPHORUS IN CHILDREN *

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In spite of the voluminous literature that has been published on the metabolism of calcium and phosphorus, little has been written in regard to the minimum requirements of these minerals for growing children. The only two papers which we were able to find dealing directly with the subject were those by Sherman and Hawley [1] and Willard and Blunt.[2] However, neither of them lowered the calcium and phosphorus intake sufficiently to obtain a negative balance. Hence the minimum requirement was not established. In our study of the metabolism of undernourished children, we have obtained a great range of values in calcium and phosphorus balance. Owing to the difficulty in making some of the children eat the calculated amount of food, the calcium and phosphorus intake often fell below the minimum requirement. The following paper was therefore compiled from results obtained in this study, supplemented with additional experiments in order to establish a definite minimum requirement.

EXPERIMENTS

The technic of feeding and collecting of specimens, as well as that of the analysis for calcium, has already been described in previous communications.[3]

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* From the Nelson Morris Memorial Institute for Medical Research of the Michael Reese Hospital, Chicago. This work is part of an investigation of the metabolism of undernourished children, supported by a fund contributed jointly by the Elizabeth McCormick Memorial Foundation, Mrs. Francis Nielson and the late Mrs. Gusta Morris Rothschild.

1. Sherman, H. C., and Hawley, E.: Calcium and Phosphorus Metabolism in Childhood, J. Biol. Chem. **53**:375 (Aug.) 1922.

2. Willard, A. C., and Blunt, K.: A Comparison of Evaporated with Pasteurized Milk as a Source of Calcium, Phosphorus and Nitrogen, J. Biol. Chem. **75**:251 (Oct.) 1927.

3. Wang, C. C.; Kaucher, M., and Frank, M.: Metabolism of Undernourished Children: IV. Calcium Metabolism, Am. J. Dis. Child. **35**:856 (May) 1928. Wang, C. C.; Witt, D. B., and Felsher, A. R.: A Comparison of the Metabolism of Some Mineral Constituents of Cow's Milk and of Breast Milk in the Same Infant, Am. J. Dis. Child. **27**:352 (April) 1924.

Phosphorus was determined according to the method described by Bell and Doisy,[4] with the following modifications: Material containing from 2 to 3 mg. of phosphorus was placed in a 300 cc. Kjeldahl flask containing 5 cc. each of sulphuric acid and nitric acid and heated until the contents were well charred. While this material was heating, nitric acid was added slowly until the mixture became clear. The heating was continued for ten minutes longer. The material was cooled, transferred quantitatively into a 100 cc. volumetric flask and made up to volume. An aliquot portion, usually 20 cc., of the solution was pipetted into another 100 cc. volumetric flask containing 40 cc. distilled water. The solution was neutralized with ammonium hydroxide and made faintly acid with dilute nitric acid. From this point, the determination was carried on according to the method described by Bell and Doisy.[4]

Thirty-eight experiments of at least one week each were conducted on eighteen children, four boys and fourteen girls. With the exception of one child, who was only 4 years old, the children were between 8 and 12 years of age. The calcium intake varied from 0.013 to 0.145 Gm. of calcium oxide per kilogram of body weight per day and the phosphorus from 0.043 to 0.204 Gm. of phosphorus pentoxide per kilogram of body weight.

COMMENT

It was found, as shown in table 1, that in every case in which the calcium intake was below 0.032 Gm. of calcium oxide per kilogram of body weight, a negative balance was obtained, and in all cases in which the intake was above this figure, the balance was positive. The calcium retention varied from 0.004 Gm. to 0.025 Gm. of calcium oxide per kilogram of body weight. Although with a low calcium intake the retention of only 0.004 Gm. and 0.007 Gm. was obtained, the remaining values showed no definite relationship between the calcium retained and the calcium ingested. With the exception of three cases, when the calcium intake was below 0.032 Gm. of calcium oxide per kilogram of body weight, a greater amount of calcium was excreted in the feces than was present in the food. No relationship was observed between the urinary calcium and the calcium intake. The calcium in the feces, however, increased steadily with the increase in food calcium. Thus, when a child was on a diet containing 0.013 Gm. of calcium oxide per kilogram of body weight per day, the calcium excreted in the feces was 0.016 Gm. per kilogram; as the food calcium was increased to 0.145 Gm., the calcium excreted was likewise increased to 0.118 Gm.

Table 2 shows that a positive phosphorus retention was obtained in all cases in which the intake of phosphorus was above 0.079 Gm. expressed as phosphorus pentoxide per kilogram of body weight, and that a negative retention resulted, when the amount of phosphorus fell below this figure. The retention varied from 0.006 to 0.045 Gm. of phosphorus pentoxide per kilogram of body weight, but the variation as in the case of calcium was independent of the phosphorus intake.

4. Bell, R. D., and Doisy, E. A.: Rapid Colorimetric Methods for the Determination of Phosphorus in Urine and Blood, J. Biol. Chem. **44**:55 (Oct.) 1920.

TABLE 1.—*Average Daily Calcium Metabolism in Children (Expressed as CaO)*

Name	Sex	Age	Weight		Calcium Intake		Calcium Output						Absorption			Retention		
							Urine		Feces		Total							
			Kg.	Variation from Normal	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Percentage of Intake	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Percentage of Intake
B. E.	F	11	30.9	— 2.8	0.404	0.013	0.182	0.006	0.489	0.016	0.671	0.022	—0.085	.....	....	—0.27	.....	....
R. W.	F	11	34.7	+ 6.1	0.490	0.014	0.146	0.004	0.538	0.016	0.684	0.020	—0.048	.....	....	—0.19	.....	....
F. J.	F	10	26.9	— 4.6	0.378	0.014	0.053	0.002	0.375	0.014	0.428	0.016	—0.003	.....	....	—0.05	.....	....
R. E.	F	10	27.1	— 5.9	0.379	0.014	0.039	0.001	0.443	0.016	0.482	0.018	—0.064	.....	....	—0.10	.....	....
F. G.	F	10	27.5	— 6.5	0.378	0.014	0.063	0.002	0.438	0.016	0.501	0.018	—0.060	.....	....	—0.12	.....	....
F. G.	F	10	22.2	—22.9	0.395	0.018	0.118	0.005	0.387	0.017	0.505	0.023	—0.008	.....	....	—0.11	.....	....
F. G.	F	10	20.1	—28.7	0.422	0.021	0.202	0.010	0.436	0.022	0.638	0.032	—0.014	.....	....	—0.22	.....	....
J. G.	M	8	23.5	— 1.3	0.487	0.021	0.082	0.003	0.416	0.018	0.498	0.021	0.071	0.003	14.6	—0.01	.....	....
A. B.	F	6	16.4	—20.4	0.336	0.022	0.052	0.003	0.415	0.025	0.467	0.028	—0.079	.....	....	—0.13	.....	....
S. W.	F	8	21.0	— 2.8	0.490	0.023	0.245	0.012	0.379	0.018	0.624	0.029	0.111	0.005	22.6	—0.13	.....	....
A. J.	F	5	17.1	— 3.9	0.443	0.026	0.123	0.007	0.353	0.021	0.476	0.028	—0.010	.....	....	—0.03	.....	....
H. K.	M	10	21.6	— 9.3	0.618	0.029	0.039	0.002	0.581	0.027	0.623	0.029	0.034	0.002	5.5	—0.01	.....	....
W. K.	M	12	28.5	— 5.6	0.911	0.032	0.132	0.005	0.673	0.024	0.804	0.028	0.238	0.008	26.1	0.11	0.004	12.1
G. C.	M	10	26.0	— 7.5	0.892	0.034	0.091	0.003	0.635	0.024	0.726	0.028	0.257	0.010	28.8	0.17	0.007	19.1
K. F.	F	8	20.2	—12.2	1.107	0.055	0.036	0.002	0.712	0.035	0.748	0.037	0.395	0.020	34.8	0.359	0.018	32.4
K. B.	F	8	20.5	— 4.7	1.151	0.056	0.061	0.003	0.720	0.035	0.781	0.038	0.431	0.021	37.4	0.37	0.018	32.1
F. B.	F	10	17.5	—14.7	0.980	0.056	0.038	0.002	0.638	0.037	0.676	0.039	0.342	0.020	34.9	0.304	0.017	30.8
E. S.	F	4	12.7	—15.9	0.730	0.058	0.029	0.002	0.393	0.031	0.422	0.033	0.337	0.027	46.2	0.308	0.024	42.2
L. B.	F	11	25.1	—20.1	1.467	0.058	0.102	0.004	1.010	0.042	1.112	0.044	0.457	0.023	31.1	0.355	0.018	24.2
W. K.	M	12	28.6	— 5.3	1.788	0.063	0.257	0.009	1.192	0.042	1.448	0.051	0.596	0.021	33.3	0.340	0.012	19.0
G. C.	M	10	26.9	— 5.3	1.813	0.067	0.099	0.004	1.342	0.050	1.441	0.054	0.471	0.018	26.0	0.372	0.014	20.5
H. K.	M	10	21.7	— 8.8	1.480	0.068	0.098	0.005	0.955	0.044	1.053	0.049	0.525	0.024	35.5	0.427	0.020	28.8
E. S.	F	4	13.7	—11.0	0.932	0.068	0.024	0.002	0.561	0.060	0.585	0.043	0.371	0.027	39.8	0.347	0.025	37.3
R. W.	F	11	35.4	+ 8.3	2.473	0.070	0.166	0.005	1.812	0.051	1.978	0.056	0.471	0.018	26.0	0.372	0.014	20.5
B. E.	F	11	31.3	— 1.6	2.371	0.076	0.185	0.006	1.719	0.055	1.904	0.061	0.652	0.020	27.5	0.467	0.015	19.7
A. B.	F	6	16.9	—18.0	1.420	0.084	0.132	0.008	0.965	0.057	1.097	0.065	0.455	0.027	32.0	0.323	0.019	22.8
A. T.	F	10	27.6	— 6.1	2.341	0.085	0.237	0.009	1.626	0.059	1.863	0.069	0.715	0.026	30.5	0.478	0.017	20.4
F. J.	F	10	27.5	— 2.5	2.371	0.086	0.089	0.003	1.751	0.064	1.840	0.067	0.620	0.023	26.1	0.531	0.019	22.4
R. G.	F	10	27.9	— 3.1	2.368	0.086	0.109	0.004	1.768	0.063	1.877	0.067	0.600	0.022	25.3	0.491	0.018	20.7
K. B.	F	8	20.6	—10.5	1.770	0.086	0.041	0.002	1.371	0.067	1.412	0.069	0.399	0.019	22.5	0.358	0.017	20.2
L. B.	F	11	25.8	—17.8	2.432	0.094	0.099	0.004	1.905	0.074	2.004	0.078	0.527	0.020	21.6	0.428	0.017	17.6
F. G.	F	10	22.7	—21.2	2.326	0.102	0.256	0.011	1.745	0.077	2.001	0.088	0.581	0.026	25.0	0.325	0.014	14.0
J. G.	M	8	23.8	± 0.0	2.534	0.106	0.141	0.006	1.903	0.080	2.044	0.085	0.631	0.027	24.9	0.490	0.021	19.3
S. W.	F	8	21.9	+ 1.4	2.473	0.113	0.376	0.017	1.846	0.084	2.222	0.102	0.627	0.028	25.4	0.251	0.012	10.1
A. J.	F	5	17.9	+ 0.6	2.033	0.114	0.211	0.012	1.593	0.089	1.804	0.101	0.440	0.025	21.6	0.229	0.013	11.3
F. G.	F	10	20.6	—27.0	2.431	0.118	0.158	0.008	1.822	0.088	1.980	0.096	0.609	0.030	25.1	0.451	0.022	18.6
K. B.	F	8	20.7	— 3.7	2.747	0.133	0.145	0.007	2.102	0.102	2.247	0.109	0.645	0.031	23.5	0.500	0.024	18.2
F. B.	F	10	17.8	—13.2	2.586	0.145	0.093	0.005	2.101	0.118	2.194	0.123	0.485	0.027	18.8	0.392	0.022	15.2

TABLE 2.—*Average Daily Phosphorus Metabolism in Children (Expressed as P_2O_5)*

Name	Sex	Age	Weight		Phosphorus Intake		Phosphorus Output						Absorption			Retention		
							Urine		Feces		Total							
			Kg.	Variation from Standard	Per 24 Hr., Gm.	Per Kg. per 24 Hrs., Gm.	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Percentage of Intake	Per 24 Hr., Gm.	Per Kg. per 24 Hr., Gm.	Percentage of Intake
R. W.	F	11	34.7	+ 6.1	1.49	0.043	1.34	0.039	0.54	0.016	1.88	0.055	0.95	0.028	63.9	—0.39	.....	....
B. E.	F	11	30.9	— 2.8	1.45	0.047	1.39	0.045	0.69	0.022	2.07	0.067	0.77	0.025	52.7	—0.62	.....	....
J. G.	M	8	23.5	— 1.3	1.17	0.050	0.90	0.038	0.48	0.021	1.38	0.059	0.69	0.029	59.2	—0.21	.....	....
F. G.	F	10	27.5	— 6.5	1.43	0.052	1.24	0.045	0.90	0.033	2.14	0.078	0.53	0.019	36.9	—0.71	.....	....
R. G.	F	10	27.1	— 5.9	1.45	0.053	0.91	0.034	0.60	0.022	1.51	0.056	0.85	0.031	58.8	—0.06	.....	....
F. J.	F	10	26.9	— 4.6	1.46	0.054	1.01	0.038	0.71	0.026	1.72	0.064	0.75	0.028	51.4	—0.20	.....	....
F. G.	F	10	22.2	—22.9	1.32	0.059	1.02	0.046	0.85	0.038	1.86	0.084	0.48	0.022	36.0	—0.54	.....	....
S. W.	F	8	21.0	— 2.8	1.32	0.063	1.16	0.055	0.48	0.023	1.64	0.078	0.84	0.040	63.5	—0.32	.....	....
A. J.	F	5	17.1	— 3.9	1.17	0.068	0.87	0.051	0.68	0.040	1.56	0.091	0.48	0.028	41.3	—0.39	.....	....
A. B.	F	6	16.4	—20.4	1.11	0.068	0.71	0.043	0.55	0.034	1.25	0.077	0.55	0.035	49.1	—0.14	.....	....
F. G.	F	10	20.1	—28.7	1.41	0.070	0.80	0.040	0.92	0.046	1.72	0.086	0.49	0.024	34.8	—0.31	.....	....
H. K.	M	10	21.6	— 9.3	1.72	0.079	1.09	0.050	0.47	0.022	1.55	0.072	1.25	0.057	72.7	0.16	0.008	9.5
G. C.	M	10	26.0	— 7.5	2.22	0.085	1.27	0.049	0.69	0.027	1.95	0.075	1.53	0.059	68.9	0.26	0.010	11.7
W. K.	M	12	28.5	— 5.6	2.47	0.087	1.28	0.045	0.80	0.028	2.08	0.073	1.67	0.059	67.6	0.39	0.014	15.8
K. B.	F	8	20.2	—12.2	2.16	0.107	1.23	0.061	0.65	0.032	1.88	0.093	1.51	0.074	70.0	0.28	0.014	13.1
L. B.	F	11	25.1	—20.1	2.70	0.107	1.53	0.061	0.78	0.031	2.30	0.091	1.92	0.077	71.3	0.40	0.016	14.8
K. B.	F	8	20.5	— 4.7	2.22	0.108	1.18	0.058	0.71	0.034	1.89	0.092	1.51	0.074	68.1	0.34	0.016	15.1
F. B.	F	10	17.5	—14.7	1.90	0.108	1.12	0.064	0.63	0.036	1.75	0.099	1.26	0.072	66.9	0.15	0.009	7.9
E. S.	F	4	12.7	—15.9	1.47	0.113	1.05	0.083	0.29	0.023	1.34	0.103	1.19	0.091	80.7	0.14	0.010	9.2
R. W.	F	11	35.4	+ 8.3	4.60	0.130	2.79	0.079	1.13	0.032	3.92	0.111	3.46	0.098	75.4	0.68	0.019	14.8
W. K.	M	12	28.6	— 5.3	4.33	0.151	2.67	0.093	1.22	0.043	3.89	0.136	3.11	0.109	71.8	0.44	0.015	10.2
B. E.	F	11	31.3	— 1.6	4.76	0.152	2.59	0.083	1.28	0.041	3.87	0.124	3.48	0.111	73.2	0.89	0.028	18.8
J. G.	M	8	23.8	± 0.0	3.76	0.158	2.02	0.085	1.14	0.047	3.16	0.132	2.62	0.110	69.7	0.60	0.025	16.0
F. G.	F	10	27.6	— 6.1	4.36	0.158	3.04	0.110	1.14	0.041	4.18	0.150	3.22	0.116	73.8	0.18	0.006	4.1
H. K.	F	10	21.7	— 8.8	3.44	0.159	2.06	0.095	1.05	0.048	3.11	0.143	2.39	0.110	69.5	0.33	0.015	9.6
G. C.	M	10	26.9	— 5.3	4.32	0.161	2.13	0.079	1.82	0.068	3.95	0.147	2.50	0.093	57.9	0.37	0.014	8.6
A. B.	F	6	16.9	—18.0	2.76	0.163	1.44	0.085	1.36	0.080	2.80	0.165	1.39	0.082	50.5	0.54	0.032	10.0
E. S.	F	4	13.7	—11.0	2.28	0.166	1.47	0.107	0.60	0.043	2.09	0.152	1.67	0.122	73.3	0.21	0.015	9.0
R. G.	F	10	27.9	— 3.1	4.74	0.170	2.44	0.087	1.92	0.069	4.36	0.156	2.82	0.102	59.6	0.38	0.014	8.0
K. Bo.	F	8	20.6	—10.5	3.54	0.172	1.91	0.093	1.33	0.065	3.24	0.158	2.21	0.107	62.4	0.30	0.015	8.5
F. J.	F	10	27.5	— 2.5	4.75	0.173	2.23	0.081	1.81	0.066	4.04	0.147	2.94	0.107	61.9	0.71	0.026	14.8
K. B.	F	8	20.7	— 3.7	3.59	0.173	1.80	0.087	1.51	0.073	3.31	0.160	2.08	0.101	57.9	0.28	0.014	7.8
F. B.	F	10	17.8	—13.2	3.16	0.178	1.59	0.089	1.15	0.065	2.74	0.154	2.01	0.113	63.6	0.42	0.024	13.3
L. B.	F	11	25.8	—17.8	4.62	0.179	2.37	0.092	1.83	0.071	4.20	0.163	2.79	0.108	60.3	0.42	0.016	9.0
F. G.	F	10	22.7	—21.2	4.35	0.192	2.65	0.117	1.15	0.051	3.80	0.167	3.20	0.141	73.6	0.55	0.024	12.6
A. J.	F	5	17.9	+ 0.6	3.38	0.193	1.79	0.100	1.37	0.077	3.16	0.177	2.00	0.122	59.4	0.21	0.012	6.3
F. G.	F	10	20.6	—27.0	4.18	0.203	1.97	0.096	1.54	0.075	3.51	0.170	2.64	0.128	63.2	0.67	0.033	16.0
S. W.	F	8	21.9	+ 1.4	4.46	0.204	2.54	0.116	0.92	0.042	3.46	0.158	3.54	0.162	79.3	0.99	0.045	22.3

As the intake was increased, an augmentation of the phosphorus was demonstrated in both urine and feces, as well as in absorption. With but one exception, the value of urinary phosphorus was greater than that of the feces, ranging from 0.034 to 0.117 Gm. of phosphorus pentoxide per kilogram of body weight, while the corresponding values for phosphorus in feces were from 0.016 to 0.077 Gm.

Based on these observations, the minimum and not the optimum calcium and phosphorus requirements for a child about 8 years of age, weighing 20 kilograms, and living on a mixed diet would be 0.64 Gm. of calcium oxide and 1.58 Gm. of phosphorus pentoxide per day in order to give a positive balance. These figures are much below Holt's [5] recommendation of one quart of milk per day, which contains approximately 1.6 Gm. of calcium oxide and 2 Gm. of phosphorus pentoxide. On the other hand, in the first series of the investigation on the calcium and phosphorus metabolism in childhood reported by Sherman and Hawley,[1] a positive balance in both calcium and phosphorus was obtained on a mixed diet containing 0.055 Gm. of calcium oxide and 0.124 Gm. of phosphorus pentoxide per kilogram of body weight. (For convenience, their figures were recomputed in terms of calcium oxide and phosphorus pentoxide.) However, the calcium and phosphorus of their diet were derived mostly from milk, the intake varying from 500 to 750 Gm. per day. In our experiments, some children consumed only 130 Gm. of milk, while others had as much as 1,400 Gm. The discrepancy in calcium intake between Sherman's results and ours is undoubtedly due in part to the difference in the source of the calcium. Since not all children drink sufficient milk to satisfy their calcium requirement, a more liberal figure should be recommended.

SUMMARY

Thirty-eight experiments of at least one week each were conducted on eighteen children with a calcium intake varying from 0.013 to 0.145 Gm. of calcium oxide per kilogram of body weight and the phosphorus intake from 0.043 to 0.204 Gm. of phosphorus pentoxide.

In every case, when the calcium intake was above 0.032 Gm. of calcium oxide and the phosphorus intake above 0.079 Gm. of phosphorus pentoxide per kilogram of body weight a positive balance was obtained. The calcium retention varied from 0.004 to 0.025 Gm. of calcium oxide per kilogram of body weight. The corresponding figures for the phosphorus were from 0.006 to 0.045.

Urinary calcium was independent of the calcium intake, while the fecal calcium increased with an increase in the calcium of the food.

5. Holt, L. F.: Food, Health and Growth, New York, The MacMillan Company, 1922.

An increased phosphorus intake resulted in an augmentation of phosphorus in both urine and feces. With but one exception, the urinary phosphorus was always greater than the fecal phosphorus.

Based on our observations, the minimum and not the optimum calcium and phosphorus requirements of an 8 year old child weighing 20 kilograms and living on a mixed diet would be 0.64 Gm. of calcium oxide and 1.58 Gm. of phosphorus pentoxide per day.

ASTHMA IN CHILDREN

IX. RÔLE OF ENVIRONMENT IN THE TREATMENT OF A SELECTED GROUP OF CASES: A PLEA FOR A "HOME" AS A RESTORATIVE MEASURE *

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With the introduction of protein skin tests in the past decade, the entire subject of asthma from the standpoint of etiology and treatment has undergone radical revision. The term "allergy" has been almost universally adopted to include asthma and other conditions exhibiting sensitization phenomena. Clinics devoted entirely to the study of allergy have been rapidly developed and are being conducted by physicians whose interests are exclusively or almost wholly confined to this subject. Finally, the need of studying allergy in children as a special class was recognized, so that now many hospitals have well organized clinics devoted to the treatment of children with asthma.

The literature is replete with communications dealing with many phases of asthma. The majority of workers in allergy stressed skin tests as the principal means of determining the exciting or causative factors of asthma, which resulted in the classification of a large group of patients as nonsensitive to protein. Later, with improved technic in the preparation of the test materials, the number of patients previously classified as nonsensitive to protein was considerably reduced. In spite of this there remained a significant number in whom histories consistent with pollen as the etiologic factor were elicited, although they remained skin insensitive (to scratch and intradermal technics). I [1] reported 7 per cent of a series of 100 children with asthma, and Kahn and Grothaus [2] 13 per cent of their 115 patients of all ages with hayfever and asthma, as belonging in this class. I suggested the ophthalmic test with pure pollen and Kahn and Grothaus the subcutaneous injection tests, thus reducing further the number of patients classified as nonsensitive. Recently, Rowe [3] and Dale and Thornberg [4] devised a system

* Submitted for publication, Dec. 5, 1929.

* From the Children's Asthma Clinic, Mount Sinai Hospital.

1. Peshkin, M. M.: Asthma in Children: I. Etiology, Am. J. Dis. Child. **31**:763 (June) 1926.

2. Kahn, I. S., and Grothaus, E. M.: Studies in Pollen Sensitiveness, M. J. & Rec. **121**:664 (June) 1925.

3. Rowe, A. H.: Food Allergy: Its Manifestations, Diagnosis and Treatment, J. A. M. A. **91**:1623 (Nov. 24) 1928.

4. Dale, Jane; and Thornberg, H. D.: Diets for the Identification of Food Allergies, J. A. M. A. **93**:505 (Aug. 17) 1929.

of elimination diets for the detection of sensitization to foods in patients still exhibiting negative skin tests.

Thus it became evident that to fix the causative factor in asthma there is required not only a complete series of protein skin tests accurately performed and properly interpreted but a carefully taken history, a physical examination, including roentgenoscopy of the chest, tuberculin skin and various laboratory tests and a careful study of the patient over a considerable period.

A practical method of classification of asthma along etiologic lines is now possible, and it is of extreme value since it leads directly to rational therapy. In this new classification there are three main groups, which include all types of asthma: (1) protein sensitive asthma, (2) protein nonsensitive asthma and (3) para-asthma.[5]

As a result of modern treatment, the percentage of children definitely improved or relieved from asthma varies widely; according to the reports in the literature, from 40 to as high as 90 per cent. This great discrepancy in results probably depends, in part, on the environment, nationality, age of onset, age when first observed, contact, potency and number of proteins employed, method of testing, interpretation of the reaction, thoroughness of the history and physical examination, method of treatment and the period of observation.

It must therefore be conceded that at least between 10 and 60 per cent of the patients who have received modern treatment continue to have asthma. Within this class of unimproved patients there exists, in my experience, a subgroup of children in whom the asthma remained severely chronic from the point of view of intensity, frequency and long duration of the attacks. It is with this group of children that this communication is concerned.

Extensive experience in treating patients with allergic diseases forces the realization that protein sensitization alone does not entirely explain

5. Patients with para-asthma (a new term described by the author in the article referred to in footnote 1) cannot clinically be distinguished from those with other types of asthma; from the point of view of etiology they must be differentiated from all other groups. These cases are directly caused by an enlarged thymus or enlarged bronchial lymph nodes or both and also by any pathologic condition inducing tracheobronchial stenosis. They are sometimes referred to as obstructive asthma. This is an inadequate term, since a majority of subjects with these abnormalities do not show asthmatic symptoms. Three patients with para-asthma, after appropriate treatment, were relieved from all symptoms for periods ranging from two to four years. At the end of these periods symptoms recurred, and the condition was diagnosed as bronchial asthma of the protein nonsensitive type because the factors responsible for the para-asthma were now absent.

the varied manifestations associated with allergy. It has been demonstrated that 10 per cent of the children with asthma whose skin was sensitive to various proteins showed no etiologic connection between protein sensitization and asthma.[1] Then again, in those patients in whom protein sensitization was found to be of etiologic importance, it was always evident that these sensitizing substances were only exciting factors and not the basic cause of symptoms. Many authors regard the basis of allergy as a disturbed balance between the sympathetic and the parasympathetic nervous systems. A more logical conception of allergy would be that there exists within the body a "physicochemical disturbance" which probably manifests itself through the nervous system.[6] If the body is in a state of "physicochemical equilibrium," the allergic patient enjoys freedom from symptoms in spite of exposure to an etiologic protein. Such a patient is said to have acquired protein tolerance. Failure to appreciate the fact that fluctuating variations of tolerance exist in different patients as well as in the same patient leads to misinterpretation of the rôle that protein sensitization plays in asthma. Vaughan [7] looks on these subjects during the intervals of freedom from symptoms as being in a "balanced allergic state."

If any factor is allowed to overthrow this governing mechanism, symptoms will result. These factors may be specific or nonspecific or both. The specific factor may be an overdose of the sensitizing protein. Overdosage by the inhalation or ingestion of sensitizing substances is too well known to require illustration. It has been observed that rabbit hair in sensitized patients may induce asthma almost immediately after exposure. Again, the symptoms may not develop until a period varying from six months to four years has elapsed, in spite of constant exposure.[1] This could be explained only by the fact that in the first instance the physicochemical equilibrium was completely unbalanced, and in the latter instances it took from six months to four years of constant irritation to induce physicochemical imbalance resulting in definite symptoms.

It has been stated that the protective mechanism which maintains the physicochemical balance may also become disturbed through nonspecific causes. Various conditions and diseases are often the determining factors in initiating the onset of asthma.[6] If exposure to the sensitizing protein is insufficient to disturb the physicochemical equilibrium, an intercurrent infection is often sufficient to produce complete imbalance.

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6. Peshkin, M. M.: Asthma in Children: III. The Incidence and Significance of Various Diseases and Infections, and of Tonsillectomy and Adenoidectomy, Am. J. Dis. Child. **33**:880 (June) 1927.

7. Vaughan, W. T.: Interaction of Specific and Nonspecific Factors in Allergic Disease, Virginia M. Monthly **51**:472 (Nov.) 1924.

However, in the absence of exposure to the sensitizing substance, an intercurrent infection will not produce asthma in the presence of a partially maintained balance.

When the physicochemical balance was profoundly disturbed, the various nonspecific and specific factors always induced asthma, in spite of the fact that the patient was receiving appropriate specific treatment at the time. In these cases there is little that can be done to give relief until nature itself completely or partially restores the physicochemical balance.

With this theory in mind, a refractory group of children with asthma was subjected to environmental treatment in an effort to restore the physicochemical balance.

The material for this study is based on forty-one cases of chronic asthma, representing approximately 10 per cent of a series of four hundred and twenty-five children, all of whom had been subjected to the accepted standards of modern investigation and treatment. These children have been under continuous observation for from two to seven years, or an average of four and two-thirds years per patient. Of the forty-one patients, ranging in age from 2 to 14 years, twenty-five (twenty-two children sensitive to protein, three nonsensitive) received the benefit of change of environment. The remaining sixteen children, because of lack of facilities, were not treated in this manner, and so indirectly served as a control series. These children are still suffering from asthma in a severe form.

Observations of clinical interest noted in this selected group of cases were as follows:

All of the children were underweight, restless and irritable and had secondary anemia. Many were undersized. Sleep, so important to health, was largely interrupted and curtailed. All of these patients had varying degrees of chronic bronchitis, and in 40 per cent of them a deformity of the chest with chronic emphysema was also present. Another 12 per cent had emphysema (volumen pulmonum auctum) without deformity of the chest.

Moving the home from one section of the city to another proved an economic hardship and a costly failure. The removal of furniture, draperies, carpets, articles of apparel and other substances, as well as carriers known to cause allergy, until in some cases the interior of the home took on the appearance of an army barrack, proved of no avail.

The floors and walls of several homes were scrubbed with various dilutions of mercuric chloride. The solution was kept in contact with the surface to which it was applied for at least one-half hour, but this

proved ineffective. The procedure was suggested by the work of van Leeuwen [8] with aspergilli.[9]

On the other hand, the removal of the child, usually in a condition of "status asthmaticus," to the ward of the hospital resulted, in the majority of instances, in complete relief from all symptoms of asthma within the first three days after admission, and freedom from asthma was maintained during the entire stay in the hospital. However, when the child was returned to its home, asthma recurred within several days and most often the first night.[10]

Twenty-five children were sent to the country for total periods of from two months to more than a year. In this new environment inhalant and dietetic restrictions were less rigid than those maintained at home. Twenty-three of these children, or 92 per cent, were markedly improved or relieved from asthma. Of the twenty-five children, eight are still in the country. The remaining seventeen children have been at home for periods varying from six months to five years, or an average of two years per patient. Thirteen of these seventeen children, or 76 per cent, are now greatly improved or relieved from asthma.

It was found that freedom from asthma following the return of the child to its home was mainly dependent on the type of patient and the period of time spent away from home. If asthma recurred in mild form, the treatment which previous to the environmental change failed

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8. Van Leeuwen, W. S.: Allergic Diseases, Philadelphia, J. B. Lippincott Company, 1925.

9. Fifty patients were tested by the intradermal method with a solution of Aspergillus fumigatus, prepared in Holland and furnished by van Leeuwen, with negative results. Another series was likewise tested with a solution of molds cultured from the wall paper of the home of one of the children, with negative results. These results, however, do not disprove the work of van Leeuwen, who concluded that in the low regions of Holland the occurrence of asthmatic and other allergic attacks was mainly dependent on the influence of the climate, and that these factors of climate were identified with a special substance (climate allergens or miasms), the nature of which is unknown, occurring in the air in the lower countries and lacking or present in a minor degree in higher altitudes. It is believed that among the substances that cause allergic diseases of climatic type, fungi, or rather products of fungi and yeast, are prevalent.

10. The pure air treatment of patients with asthma in miasm-proof chambers was introduced by van Leeuwen in 1924 (Bronchial Asthma in Relation to Climate, Proc. Roy. Soc. Med., Sect. Therap. and Pharm. **17**:19 [April] 1924). Such treatment for patients residing in Holland has proved successful. The children with chronic asthma in my series were not treated in this manner, because the construction of such chambers in their homes was economically unpractical. The main causes for the asthma in my cases were dependent not on influences of climate, but rather on those of environment. The homes of all these children were within five miles of the hospital and in several instances only a few hundred feet.

to bring about any improvement now usually proved efficacious. Frequently, however, children had to be returned to the favorable environment two or more times before the tendency toward recurrence of asthma in a severe form was controlled. In most of the latter cases, absence from home for any one period did not exceed two months, the reason for this being that most institutions refused to maintain a child with asthma for longer periods. Often it was difficult to keep these children even for that length of time in any one institution, so that they had to be shifted from one convalescent home to another. This problem has become more and more difficult and now most of these institutions refuse to admit such children. What is then to be done with them? In the light of this experience, it would appear that the most logical and humane solution would be the creation of homes especially adapted to the needs of this type of asthmatic child. The social service department of Mount Sinai Hospital has recognized this need and has recommended the establishment of such a home to take care of those children attending the allergy clinic who are in need of environmental treatment.

COMMENT

The clinical significance of the protein skin reactions is far from a closed chapter. After all, they are of value only when they lead to direct clinical results. There are many workers who consider the present status of the question as eminently satisfactory and almost a complete science. Among these workers are some who contend that no definitely allergic patient need resort to change of environment for relief from asthma. In reply to this contention, the simple example can be cited of the patient with seasonal asthma due to ragweed sensitization in whom no nasal pathologic condition or other disturbance exists. In spite of a well regulated and administered course of treatment, the patient continues to have asthma. The only measure of relief is found when the patient removes himself to an environment artificially or naturally free from pollen. This type of patient is not uncommon in the experience of most authoritative workers, and this refractory characteristic occurs in cases of asthma in which the exciting causes are other than pollen.

It appears reasonable to assume that in this group of children the principal exciting factor of asthma must be in or about their homes. In spite of modern and intensive investigation, these factors still remain unknown. The majority of these children were sensitive to protein, and specific or nonspecific therapy left the asthma unchanged. The contention may be advanced that house dust is the major etiologic factor of asthma, but failure to bring about relief by specific treatment is due to causes similar to those encountered in the case of pollen sensitization

cited. However, if this contention is acceptable, it carries with it an admission of the futility of further specific treatment. The child with chronic asthma is therefore consigned to the fate of "outgrowing" its affliction. The other alternative is environmental treatment, which has proved of definite value in this study.

CONCLUSIONS

1. In a series of four hundred and twenty-five cases of asthma, it was found that in forty-one children, ranging in age from 2 to 14 years, or approximately 10 per cent, the asthma remained severe, persistent and of long duration, in spite of intensive modern treatment.

2. Twenty-five children (twenty-two children sensitive and three nonsensitive to protein) were treated by a change of environment. In spite of the fact that the inhalant and the dietetic restrictions were kept less rigid than at home, twenty-three children, or 92 per cent, were markedly improved or entirely relieved of asthma. Seventeen of these twenty-five children have been returned to their homes for periods varying from six months to five years or an average of two years per patient. Thirteen of these seventeen children, or 76 per cent, are now greatly improved or relieved from asthma. In some of these children appropriate treatment after change of environment was successful, whereas it had failed before.

3. Sixteen of these forty-one children were not treated by a change of environment because of lack of facilities and other reasons and so indirectly served as a control series. These children are still suffering from chronic asthma.

4. Allergy per se cannot be explained entirely on a basis of protein sensitization, because the sensitizing substances in themselves are merely exciting factors and not the basic cause of the symptoms. A patient in a state of "physicochemical equilibrium" enjoys freedom from symptoms in spite of exposure to etiologic substances. If any factor overthrows this governing mechanism, symptoms will appear. These factors may be specific or nonspecific or both. An appreciation of this fact will aid in establishing a clearer conception of asthma and will lay the foundation for a more intelligent management of the disease in childhood.

5. When the physicochemical imbalance was profoundly disturbed, the various specific and nonspecific factors always induced asthma, in spite of the fact that the patient was receiving appropriate treatment at the time. In these cases there is little that can be done to give relief until nature itself restores the physicochemical balance. In these cases it has been found that a change of environment, preferably to a "home" prepared to accommodate allergic patients, was of definite value in partially or completely restoring physicochemical balance.

6. Until newer methods of treatment are advanced which will successfully control or free this group of children from asthma, the establishment of a "home" where a child with chronic refractory asthma can be kept for at least six months is regarded as a humane, urgent and economic necessity as well as a therapeutic measure of definite value.

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THYMIC SHADOWS OF NEW-BORN INFANTS*

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Considerable interest has been recently aroused in regard to the so-called thymic shadow in children. Our interest has been centered particularly about the thymic shadow in the new-born infant. With this in mind, we studied 100 consecutive new-born infants at Mount Sinai Hospital to determine, if possible, the frequency with which these shadows were observed and also their association with the so-called cardinal symptoms. The thymus has been widely discussed since the time of Plater, who in 1662 described the first case of "thymico-lymphatic death." Especially has interest been aroused during the past ten years. Nearly everything associated with respiratory difficulties has been attributed to enlargement of the thymus. Morse [1] in a recent paper said, "It has become the fashion of physicians to attribute all disturbances of infants not definitely associated with rickets to the thymus. This is particularly true when the x-ray shows what apparently is an enlargement of the gland."

Just how large a thymus must be to produce symptoms of obstruction is a question. Finkelstein stated that he had never seen a case of stenosis due to thymic compression. Kleinschmidt [2] stated that many cases so diagnosed have been proved by autopsies to be otherwise. Birk and Scholl, in a recent series of 1,000 infants, found actual hyperplasia in about four. In a survey of the Benjamin Clinic at Cincinnati, Friedlander [3] reported that 8 per cent of all babies had abnormally large thymuses. Bonar,[4] in a recent series of new-born infants, showed that in about 40 per cent there were radiologic evidences of enlargement of the thymus without any apparent symptoms. Liss,[5] in a series of 119 infants, showed roentgenologic evidence of enlargement in 42 per cent.

* Submitted for publication, Nov. 23, 1929.

* From the Departments of Roentgenology and Pediatrics, Marquette University Medical School and Mount Sinai Hospital.

1. Morse, J. L.: Thymus Obsession, Boston, M. & S. J. **197**:1547 (Feb. 16) 1928.

2. Kleinschmidt, H.: Monatschr. f. Kinderh. **37**:358, 1927.

3. Friedlander, A.: Progress in Pediatrics: The Diagnosis and Treatment of Enlarged Thymus, Am. J. Dis. Child. **6**:38 (July) 1913.

4. Bonar, B. C.: Northwest Med. **27**:178 (April) 1928.

5. Liss, I. E.: Thymic Shadows, Am. J. Dis. Child. **24**:192 (Sept.) 1922.

Because of the many variations of technic and of interpretation, it has been difficult to establish a satisfactory concept of a normal thymic shadow. Wasson,[6] with an elaborate roentgenographic technic, was perhaps the first to establish what could definitely be called a normal thymic shadow. Wasson made x-ray pictures of both living patients and cadavers. Wires and various opaque objects were placed about the thoracic viscera and left in situ, and x-ray pictures were taken to determine what relationship existed between their actual size and the x-ray shadows. The following deductions were made:

At birth, the shadow usually attributed to the heart is that of the heart. The right border is formed by the superior and inferior vena cava. The right ventricle lies to the left of the right border and the

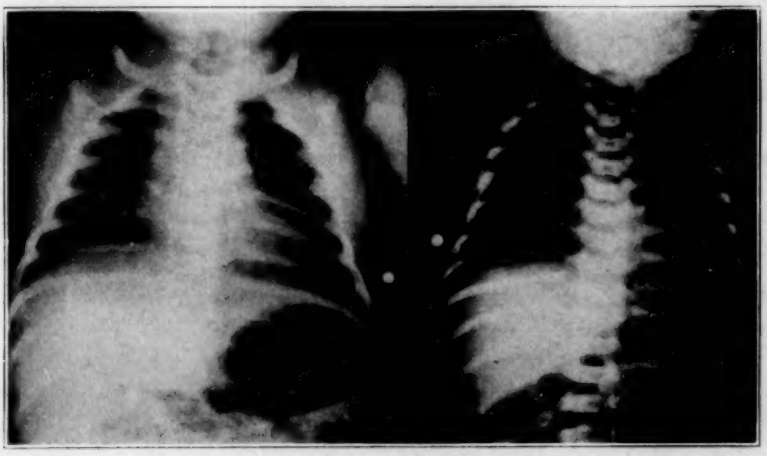

Fig. 1.—The upper and lower limits of what is considered normal in shadows of thymic glands.

right auricle above to the left. The left border of the heart is formed by the left ventricle. The two auricles and the great vessels form the base of the heart shadow. The superior vena cava forms the right border of the great vessels' shadow as they come off from the heart. The pulmonary artery is found on the left border, and it quickly divides into the right and left pulmonary arteries. The aorta lies in the middle of the mediastinum, and in the new-born infant is not seen as a distinct shadow because of its position. It passes directly upward and turns directly backward, passing down beyond the heart. The aorta and the pulmonary artery are nearly of the same size at birth. If there is no appreciable enlargement of the thymus present, these great vessels, that is, the vena cava, the aorta and the pulmonary artery, form a

6. Wasson, W. W.: Thymus Gland, Arch. Otolaryng. **4**:495 (Dec.) 1926.

shadow, triangular in shape, which quickly narrows as it passes upward toward the first rib. At its narrowest point, its width is less than the transverse diameter of the shadow of the spinal column. The trachea with its air column is observed to pass down in the midline beyond the aorta and to bifurcate between the third and fifth thoracic vertebrae into its right and left main branches. The esophagus lies beyond the trachea, but is not usually discernible. If the thymus is at all enlarged, it is found to overlap the base of the heart and to pass upward, obscuring the shadow of the great vessels just described. The shadow of the thymus is also triangular, with its base overlapping the base of the heart, especially the auricles. Its thoracic diameter at its broadest point, which is usually the third interspace, may be from two to three times the diameter of the shadow caused by the thoracic spine. This shadow of the spine is taken by measuring the transverse diameter of the vertebral bodies. If the shadow of the thymus is no greater than that caused by the great vessels, the thymus is small. If, however, the shadow is wider than that caused by the great vessels, the thymus is considered large.

The thymus lies anteriorly across the upper part of the sternum or manubrium and posteriorly on the auricle, the great vessels and the trachea. Laterally, it is overlapped by the anterior margin of the lungs and when fairly large pushes the lungs to either side; thus, it will be noted that the thymus lies between the bony structure anteriorly and the bony column posteriorly, with the great vessels and the trachea between the thymus and this bony posterior spine. Only laterally and downward has it any chance to spread during its growth. The technic used in these observations is of great importance, inasmuch as distance, position and phases of respiration play an important part. Great variation has been found in the size of the thymus between the recumbent and the upright positions, and between the inspiratory and expiratory phases of respiration. It has been found that the so-called postero-anterior position in recumbency at full inspiration has been best. The diaphragm must curve downward from well beneath the heart and its crest should maintain a uniform level on the thoracic spine, which is usually about the eighth vertebra. Under these conditions, the mediastinal viscera occupy the mediastinal spaces with ease and neither produce unnatural bulging of the thymus nor create a space into which the thymus may fall. In other words, the mediastinal shadows under these conditions, when measured at the level of the second thoracic interspace, is about one and a third times the bodies of the thoracic vertebrae, unless it is increased by the thymus or by some pathologic condition.

The variation in the size of the thymus between inspiration and expiration has been noted many times, particularly by Kleinschmidt,[2] Wasson,[7] Alexander,[8] Gerstenberger,[9] King,[10] Bonar,[4] Podlasky, Morse [1] and others. In all, it has been found that the shadow becomes narrower on inspiration and broader on expiration.

OBSERVATIONS

X-ray pictures were taken of the chests of one hundred consecutive new-born infants during the first twenty-four hours of life. Although an effort was made, it was not always possible to obtain these pictures in the same phases of respiration. However, the technic remained constant, that is, a distance of 30 inches (76.20 cm.), one-eight of a second for the time of exposure, at 25 milliamperes and 55,000 volts. Of the one hundred infants, thirty-five showed definitely enlarged thymic shadows; that is, the width at the second thoracic vertebra proved to be more than twice that of the latter's transverse diameter.

Whether the month of birth has any relation to size could not be ascertained as all the infants studied were born during the months from July to and through November. However, August and October showed the greatest number, with ten each; July showed seven; November, five, and September, three (table, *a*).

Sex apparently did not play an important rôle. Sixteen were female and nineteen male.

The birth weight seems to have some definite bearing on the size of the gland, the heaviest infant perhaps having the largest gland. Of the thirty-five infants, one weighed 5 pounds (2.3 Kg.); seven, 6 pounds (2.7 Kg.); eleven, 7 pounds (3.2 Kg.); ten, 8 pounds (3.6 Kg.), and six, 9 pounds (4.1 Kg.) (table, *b*).

Whether position in utero and difficulties in deliveries have any bearing is a question. At any rate, the thirty-five infants with large thymus presented as follows: thirteen left occipital anterior, nine right occipital anterior, three left occipital posterior, four right occipital posterior, three breech and three not stated (table, *c*). Most of the anterior positions represented normal or low forceps deliveries.

The cry at birth, in spite of the fact that the mother was either partially or completely anesthetized, was vigorous in twenty-six of the thirty-five infants; in two, it was fair, but prompt; in three it was poor,

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7. Wasson, Walter: Thymic Stridor, J. A. M. A. **89**:1025 (Sept. 24) 1927.

8 Alexander, William: X-ray in Children, Wisconsin M. J. **26**:441 (Sept.) 1927.

9. Gerstenberger, H. J.: Factor of Position of Diaphragm in Roentgen-Ray Diagnosis of Enlarged Thymus, Am. J. Dis. Child. **21**:534 (June) 1921.

10. King, J.: Radiology **9**:148 (Aug.) 1927.

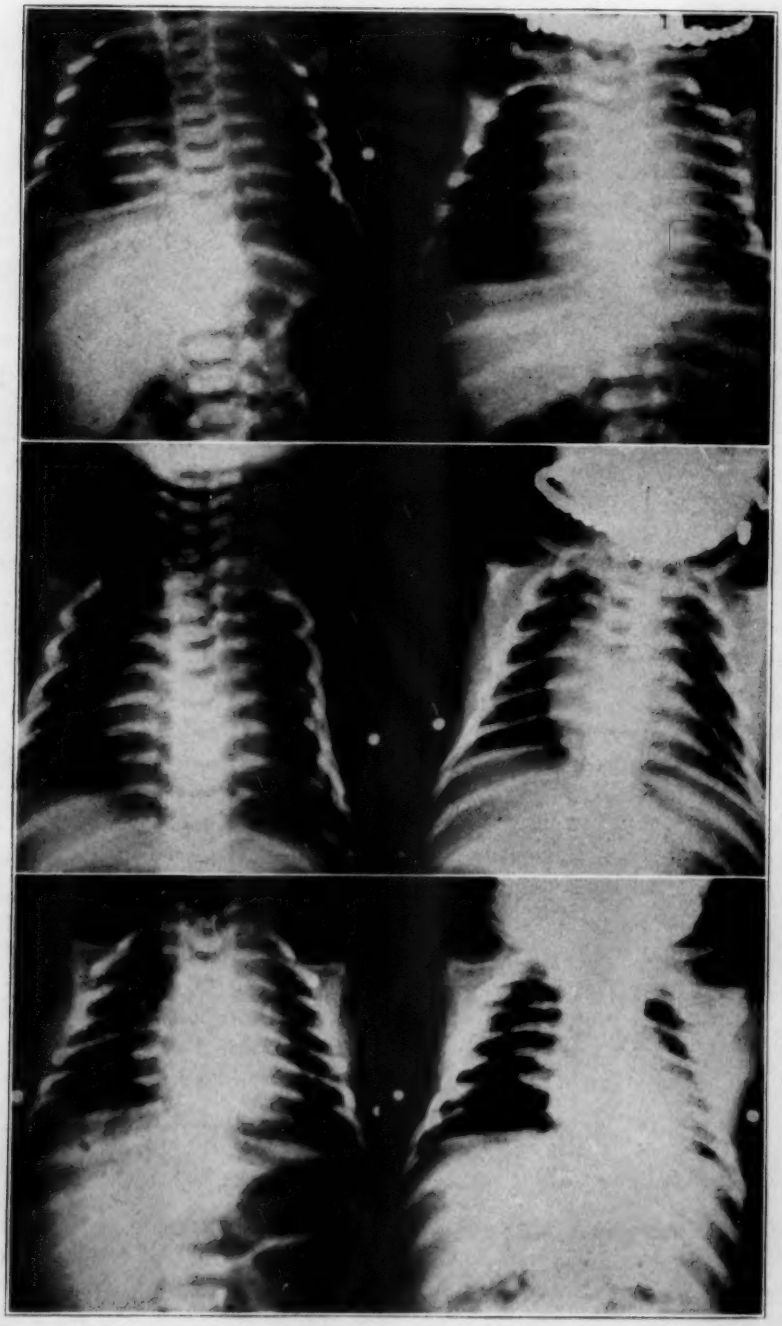

Fig. 2.—Shadows of enlarged thymic glands.

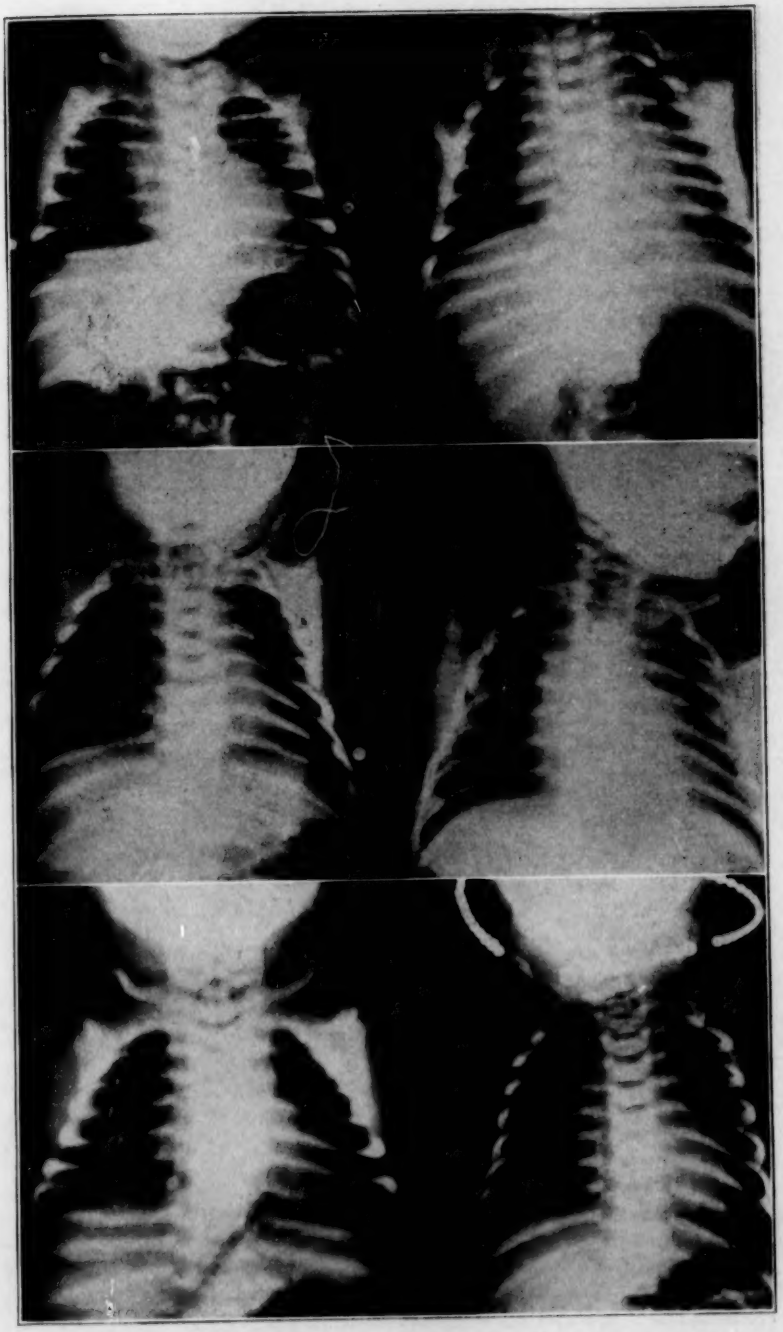

Fig. 3.—Shadows of enlarged thymic glands.

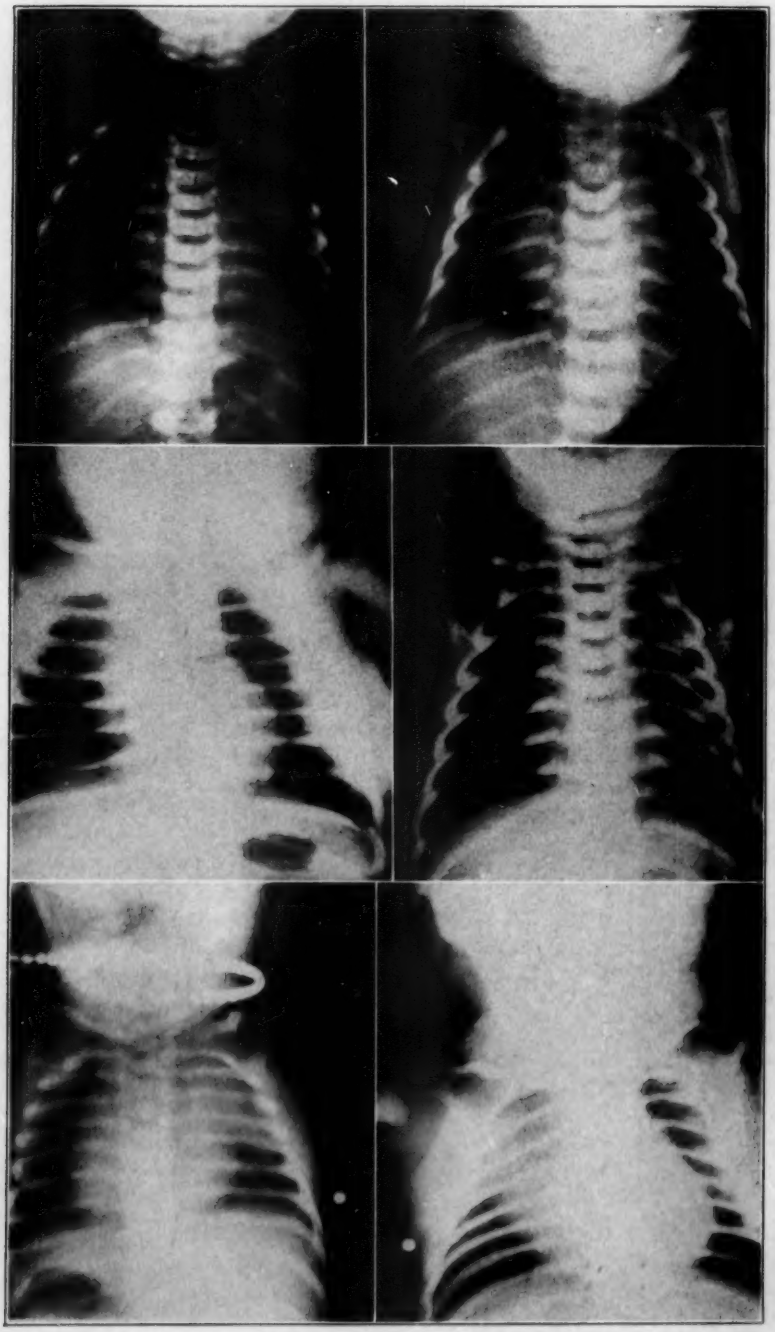

Fig. 4.—Shadows of enlarged thymic glands.

and in only four was it delayed, and in these instances it was associated with difficulty in resuscitation. Cyanosis was noted in only two infants; in one it was marked and in one, rather slight. In neither instance was it associated with stridor, and in neither instance did it persist (table, *d*).

A stridor was noted in but one infant, and was not associated with any other symptom suggestive of thymic enlargement.

Twenty-nine of the thirty-five infants took food well after the first day (table, *e*); three, fairly well, and three, rather poorly. However,

Observations on Infants with Large Thymuses

(*a*) Month of Birth

July	August	September	October	November
7	10	3	10	5

(*b*) Birth Weight

5 Pounds	6 Pounds	7 Pounds	8 Pounds	9 Pounds
1	7	11	10	9

(*c*) Position in Utero

Left Occipital Anterior	Right Occipital Anterior	Left Occipital Posterior	Right Occipital Posterior	Breech	Not Stated
13	9	3	4	3	3

(*d*) Cry at Birth

Vigorous	Fair	Poor	Delayed
26	2	3	4

(*e*) Feeding after First Day

Well	Fairly	Poorly
29	3	3

(*f*) Primary Rise in Weight

Second Day	Third Day	Fourth Day	Fifth Day	Not Stated
4	12	11	2	6

the primary rise in weight was noted on the second day in four, on the third day in twelve, on the fourth day in eleven and on the fifth day in two, showing little or no diversion from the normal (table, *f*). In every instance, the child was active and alert and was discharged on or about the tenth day with all evidences of being a normal infant.

SUMMARY

One hundred consecutive new-born infants were studied. Thirty-five showed definitely enlarged thymuses. None of the symptoms usually referable to thymic enlargement was evident in the thirty-five described. All infants were discharged from the hospital in good condition.

Case Reports

CAVERNOUS MALFORMATION ABOUT THE PORTAL VEIN MANIFESTED CLINICALLY AS A CHRONIC THROMBOSIS OF THE SPLENIC VEIN

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The clinical picture of chronic thrombosis of the splenic vein is regarded as rare, and causes great difficulty both from a diagnostic and from a therapeutic point of view. In recent years, it has become clear that the cause of the obstruction to the venous return from the spleen in this clinical condition is not always in the splenic vein itself.

This is shown also in the case reported in this article. We believe that a detailed description of this case will give greater insight into the etiology of the condition. Further, there has recently been exhibited an increased interest in pediatric circles in clinical picture, as may be deduced from the recent and extensive publication of Wallgren [1] and of Smith and Howard,[2] among others.

REPORT OF CASE

History.—At the end of February, 1926, a boy, aged 4 years, was brought to one of us on account of enlargement of the spleen. His complaints were: polyuria and pollakiuria; he was weak, pale and easily fatigued. As early as at the age of 6 months, his abdomen was distended. There were no symptoms from the stomach or intestines. There had never been jaundice, or bleeding from the nose, mouth, stomach or intestine; the urine was not hemorrhagic. Urticaria was often present. Occasionally, the temperature was subfebrile.

Examination.—The infant was born at full term, and at birth there was no question of an umbilical infection. At the first examination, we found that he was intelligent, with no signs of infantilism. He looked pale, and slightly yellow.

* Submitted for publication, Oct. 22, 1929.

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1. Wallgren, A.: Contribution à l'étude des splénomégalies de l'enfance, Acta paediat. **6**:1, 1927.

2. Smith, R. M., and Howard, P. J.: The Early Occurrence of Gastric Hemorrhage in Children with Splenomegaly, Am. J. Dis. Child. **34**:585 (Oct.) 1927.

There was no edema; the glands were not palpable. The mucous membranes were fairly anemic. A fine net of veins was visible over the thorax, continuing over the abdomen, especially at the right side. The heart and lungs were normal. The abdomen was distended, especially in the left upper quadrant. There was no ascites. The umbilicus was not prominent and looked normal. The liver was slightly palpable under the right costal arch. In the left upper quadrant of the abdomen an enormously enlarged spleen could be felt. The size of the spleen is shown in figure 1. The disorders of micturition were explained by the pressure of the enlarged spleen on the urinary organs. Rectal examination showed nothing of importance. The fundus oculi were normal.

Examination of the blood was made repeatedly during the illness. At the first examination, a slight secondary anemia was found, but without any pathologic change in the red blood corpuscles. There were no malaria parasites in the blood. A distinct leukopenia existed (1,650 leukocytes). At the first examination, the differential count of the white cells gave the following results: polymorphonuclear neutrophils with segmented nuclei, 35; with rod-shaped nuclei, 11; eosinophils, 1; lymphocytes, 43; monocytes, 10.

Fig. 1.—Size of the spleen in comparison to that of a normal spleen.

Thus, with the exception of a small shift to the left and a relatively small lymphocytosis and monocytosis, the leukocyte picture was normal. This examination gave no evidence of disease of the blood.

The fragility test gave normal results. The reaction to the direct van den Bergh test was negative, and to the indirect test only slightly positive. There was no hematin in the serum. The reactions to the Wassermann and Sachs-Georgi tests were negative, as was the reaction to the Pirquet test. There were no worm eggs in the stools, and macroscopically there was no blood. The spectroscopic examination of the stools for occult blood sometimes gave positive results; hemochromogen and hematoporphyrin were also sometimes present. As Ratnoff [3] observed that occult blood may be present in the stools of normal children under the age of 6 years—an observation that we could confirm [4]—no par-

3. Ratnoff, H. L.: Ueber das Vorkommen von okkultem Blut in den Faeces bei Säuglingen und im frühen Kindesalter, Ztschr. f. Kinderh. **34**:340, 1923.

4. Snapper, I., and van Creveld, S.: Ueber okkulte Blutungen, Ergebn. d. inn. Med. u. Kinderh. **32**:44, 1927; Nederl. Tijdschr. v. Geneesk. **2**:401 and 525, 1927.

ticular significance could be ascribed to this fact. Symptoms of ulcus ventriculi or duodeni or of the persistence of a Meckel's diverticulum, conditions which are rare in childhood, were absent.

The history of the case did not show signs of a preceding hemorrhage of the stomach or of the intestine. Some weeks after the first examination, the child may have had tar-colored stools, but at an examination of the stools some days later, scarcely any occult blood could be demonstrated.

Diagnosis.—The diagnosis was difficult, the only definite symptoms being a very large spleen and a moderate secondary anemia. Several possibilities had to be considered. We thought that cirrhosis of the liver could be excluded as urobilin was absent from the urine. The liver was not enlarged, its consistency was normal and there was no ascites. Syphilis, tuberculosis, malaria and a disease of the blood could also be excluded.

We thought chiefly of Gaucher's disease or one of the analogous conditions which were summarized some years ago by Pick.[5] For further evidence that would lead to the diagnosis, we made a roentgen examination of the body. We did this especially because in recent years facts have been published about the occurrence of bone islands in Gaucher's disease.[6] From a diagnostic point of view, it is important that this has been found not only at autopsy, but also on roentgen examination in vivo.[7] At a thorough roentgenologic examination of different parts of the body of the patient, no changes were found. Puncture of the spleen or examination of the bone-marrow, which might also have helped in making a diagnosis of Gaucher's disease or of Pick-Niemann's disease, was thought to be dangerous. Puncture of the spleen was considered especially dangerous as there was a possibility of a circumscribed formation of congested cavities in this organ.

In order to confirm the diagnosis of Gaucher's disease, we also thought of making use of the recent investigations on the nature of the substance stored in the Gaucher cells;[8] this is kerasin. It was possible, although it had not been investigated previously, that an extensive formation of such a substance might also give rise to an excretion of this substance in the urine. We therefore examined the sediment in a large quantity of the urine of our patient for one of the characteristic properties given for the Gaucher's substance, e. g., a gelatinous precipitation with sublimate after extraction with alcohol. In our case, however, no precipitation was obtained.

In the meantime, we performed some tests for liver function (galactose, 20 Gm.; fructose, 20 Gm.), without finding any disturbance of that organ.

Course.—The patient was discharged from the clinic and remained under our control. During this time, he had no special complaints. The yellowish color remained. The abdomen was distended as always; the size of the spleen remained about the same. The urine was repeatedly examined, but showed no abnormalities, especially no urobilin or urobilinogen.

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5. Pick, L.: Der Morbus Gaucher und die ihm ähnlichen Erkrankungen, Ergebn. d. inn. Med. u. Kinderh. **29**:519, 1926.

6. Pick, L., and Zadek, J.: Morbus Gaucher, Med. Klin. **20**:78, 1924.

7. Junghagen, S.: Röntgenologische Skelettveränderungen bei Morbus Gaucher, Acta Radiol. **5**:506, 1926.

8. Lieb, H.: Ztschr. f. phys. Chem. **140**:305, 1927. Epstein, E.: Beitrag zur Pathologie der Gaucherschen Krankheit, Virchows Arch. f. path. Anat. **253**:15. 1924; Biochem. Ztschr. **145**:398, 1924.

Subsequently, repeated examination of the blood always showed a severe leukopenia (2,800; 2,200); the hemoglobin content remained about the same (± 50). The number of erythrocytes increased from 3,000,000 to 3,500,000; the number of thrombocytes was about 60,000. Occasionally, the patient had a slight fever, lasting for a few days.

In November, 1926, he was paler. He had a rise in temperature (rectally up to 39.8 C. [103.6 F.]). The abdomen grew more distended. The left upper quadrant of the abdomen was painful to palpation. The spleen was as large as before. The urine was normal. Some days later the stools could be examined, but scarcely any occult blood could be found. The hemoglobin was still 50 per cent.

On December 22, in the night, the patient suddenly had a severe hemorrhage from the stomach, the first time, as far as we know, that a hemorrhage has occurred in a patient 4 years old. There was no coughing or vomiting. After this bleeding, the abdomen was painful to the touch; the spleen could not be palpated directly. The boy was very pale.

At once we questioned whether the diagnosis of Gaucher's disease could be maintained. Hemorrhage rarely occurs in Gaucher's disease; but there are a number of diseases in children which may give rise to sudden and severe gastric bleeding.

The next day the child was again taken to the hospital. At that time the abdomen was palpable; the spleen was again very much enlarged. The gastric bleeding soon recurred a few times, and it was clear that the blood was vomited. The general condition grew worse; diarrhea developed, and the stools were mixed with much blood. Now we ascertained an important fact which caused us to abandon the diagnosis of Gaucher's disease and to make another diagnosis with more certainty. We observed occasionally that immediately after a severe hemorrhage the spleen decreased greatly in size, but soon afterward was very much enlarged again. This fact seemed to indicate with certainty that we had to do with an impediment to the venous return from the spleen, and that the bleeding from the stomach or the esophagus made possible the discharge of the splenic venous blood and of that of the engorged spleen. The clinical picture now became clear; we were dealing with a chronic thrombosis of the splenic vein. The symptoms given as characteristic for this disease, namely, a very much enlarged spleen, anemia and repeated gastric hemorrhages, were present.

After the recognition of the clinical picture, we tried to improve the patient's condition in order to remove the spleen afterward. We thought that in this way we could save his life, as it seemed that a thrombosis of the portal vein did not as yet exist, for ascites failed to appear originally, and the liver function was normal. For this reason we performed some blood transfusions at intervals; they were successful, but the gastric hemorrhages continued, and sometimes the stools also contained a good deal of blood. The patient was sinking fast; edema, ascites and strong urobilinuria also appeared, and the enlarged veins on the abdominal wall increased. On Feb. 15, 1927, the patient died.

We thought that in this case we had to do with a thrombosis of the splenic vein, which, in view of the final course of the case, had continued into the portal vein. Autopsy was performed, and gave the following results:

Autopsy.—When the abdomen was opened, clear yellow fluid appeared; altogether, about 1,250 cc. of this fluid was found in the abdominal cavity. The liver extended 1 fingerbreadth below the costal border. The spleen was greatly

enlarged and extended 4 fingerbreadths below the costal border. It weighed 1,070 Gm. An infarct of about 1 cm. in diameter was visible on the surface of the spleen.

The appendix did not show adhesions. The lymph glands in the mesentery were not enlarged. Macroscopically, nothing of importance was noted in the other abdominal organs.

No abnormalities were found in the thorax.

In the stomach, coagulated blood was found, which formed a cast of the stomach. The esophagus, stomach and duodenum were cut as usual. Many large veins were visible under the mucosa of the esophagus and stomach. Along the small curvature of the stomach there were many enlarged veins, which continued into the ligamentum hepatogastricum. About 1 cm. from the cardia a ruptured varix of the distended vena gastrica dextra was found.

The trunk of the portal vein and also the vena mesenterica superior and the splenic vein were not thrombosed, and the latter two vessels joined to form the trunk of the portal vein in a normal way. However, it was impossible to explore with a sound both the left and the right intrahepatic branches of the portal vein from the trunk. Therefore, a section of the liver was made at about the point of entrance of the portal vein into the liver. The liver was divided by this section into an anterior and a posterior part. At this section, there appeared to be cavernous tissue at the place of the porta hepatis and in the surrounding tissue, which continued in several directions into the liver tissue. On close examination, it appeared that from the point of union of the vena mesenterica superior and the splenic vein the trunk of the portal vein first extended for about 2 cm. in the direction of the liver. Then the trunk divided into two branches; the first was a thin branch with a thick wall going to the liver as the continuation of the trunk of the portal vein. This vessel was about 2 cm. long, and the lumen admitted only a small sound to a distance of 1 cm., the vessel being obliterated beyond that. The second branch extended first to the right and then a little backward. After a course of about 1 cm., it divided into two branches. Of these, one (*GC*, fig. 2) with a diameter of 8 mm. went to the right and then upward until it reached the gallbladder, and there it divided into a number of small veins. This branch, therefore, was the enormously dilated vena cystica. The other branch (*LC*, fig. 2) ran almost parallel with the trunk of the portal vein to the liver, and it could still be explored with a sound into the cavernous tissue for a distance of about 3 cm. without showing division into right and left branches and further regular branching. The course of the different blood vessels mentioned is demonstrated in figure 2. Undoubtedly, the thick walled vein with the narrow lumen just mentioned was the portal vein, and the other vessels were collateral vessels.

At the posterior part of the cut surface of the liver (fig. 3), on the border of the cavernous tissue, and on the left as well as on the right side, a blood vessel was visible as a wide opening. A bent sound could easily be passed from one opening to the other, and the blood vessels appeared to be surrounded by cavernous tissue. However, the same communication could not be demonstrated to exist between the two openings and the blood vessel going from the trunk of the portal vein to the liver, just mentioned (figure 2, *LC*). It was clear that the two openings that could be explored with a sound at the cut surface of the liver were sections through the right and the left branch of the portal vein.

A section was made of the ligamentum hepatoduodenale at a distance of 5 cm. from the point of union of the splenic and superior mesenteric veins in the direction of the liver. From the section obtained, the small blood vessel which we

thought was a continuation of the portal vein, and which, as previously mentioned, could be explored with a small sound for 1 cm. was found totally closed. It was situated just behind the arteria hepatica and continued as a small solid cord in the direction of the liver. In order to examine the continuation of this obliterated trunk of the portal vein in the liver, a piece of tissue 4 by 2 by 2 cm., containing the place of entrance of the trunk of the portal vein into the liver, was cut from the liver. This small piece of tissue was embedded in colloidin, and serial sections of it were investigated microscopically. The sections were perpendicular to the direction of the portal vein.

In the first section of this series, it appeared that the lumen of the portal vein was totally obliterated. The obliterated portal vein was situated just above

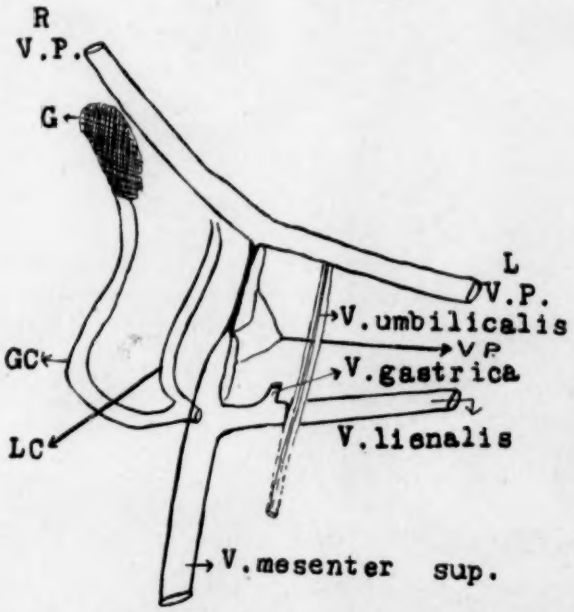

Fig. 2.—Course of the different blood vessels. *LC* indicates a collateral blood vessel; *GC*, the vena cystica. *G* indicates the gallbladder; *VP*, the portal vein; *RVP*, the right branch of the portal vein; *LVP*, the left branch of the portal vein.

the arteria hepatica (normally it lies behind this artery). At the left side, we also found a section of an artery, the lumen of which was filled for the greater part with a thrombus substance. At the lower side of the preparation, a section of a large vein was visible, a collateral blood vessel indicated in figure 2 by *LC*. As far as it was still present in the section, the liver tissue showed nothing particular. Between the several sections of the blood vessels, some fatty tissue could still be found, with small and large branches of nerves. Numerous sections of small vessels without the cavernous tissue had already developed here.

A further examination of the sections showed that the closed portal vein slowly regained its lumen, which became wider and finally continued into a

larger vein (section 320, fig. 4). Here the portal vein split into its two intrahepatic branches. Figure 4 shows sections 1, 50, 100, 150, 200, 250, 300, 320, 350 and 450. However, we found that in the first few sections the portal vein was surrounded by some sections of blood vessels. In the further study of the serial sections, the number of sections of blood vessels, as well as the cross-sectional area of each separate section, increased. Apparently, then, the so-called

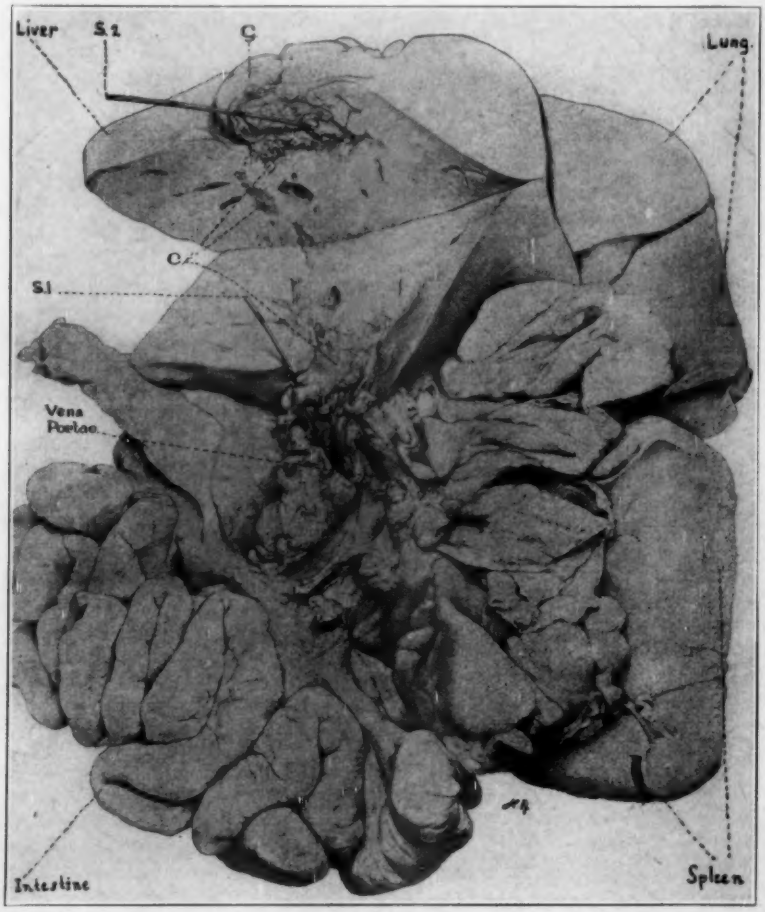

Fig. 3.—Section, showing the cut surface of the liver. *G* indicates the gallbladder; *C*, cavernous tissue in the liver; *S 1*, a sound in a collateral blood vessel to the gallbladder; *S 2*, a sound in one of the collaterals of the cavernoma.

cavernous tissue began here; it became more distinct in the further sections, in which it was found to be present also in the liver tissue.

In order to study also the continuation of the umbilical vein into the left branch of the portal vein, a second piece of liver tissue, 2.5 by 1.5 by 2 cm., was taken. This piece was also embedded in colloidin, and the serial sections of it were examined. In the first section of this series, one could see with the naked eye that the umbilical vein still existed as such. The wall was thick, and the

lumen was distinct but narrow. It further appeared that the lumen of this vessel was surrounded by a layer of endothelial cells. In continuing the serial sections further in the direction of the liver, it appeared that the lumen of the umbilical vein became gradually wider. Finally, it could easily be seen that the umbilical vein passed into the left branch of the portal vein; the lumen of both vessels was empty.

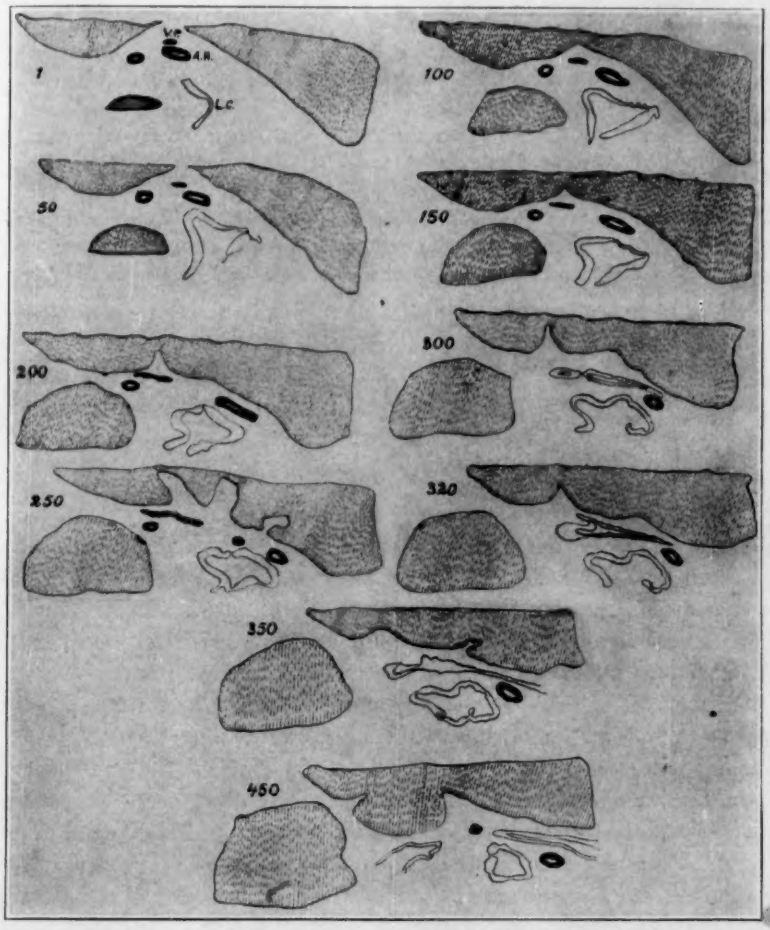

Fig. 4.—Survey of the serial sections, 1, 50, 100, 150, 200, 250, 300, 320, 350, 450.

Finally, a piece from the part of the umbilical vein lying between the umbilicus and the liver was examined microscopically. Here also it appeared that the umbilical vein had a thick wall and a distinct lumen.

Summary.—The observations in the case may be summarized as follows:

Clinical Data: 1. Great enlargement of the spleen in a boy, aged 4 years, probably existing since a short time after birth, and not combined with a perceptible enlargement or change of the function of the liver.

2. Normal physical and psychic development.

3. Fairly marked secondary anemia with marked leukopenia and relative lymphocytosis.

4. Severe hemorrhages from the stomach or esophagus without known cause. It appeared that after each severe hemorrhage the spleen had decreased much in size.

5. Diagnosis of chronic thrombosis of the splenic vein.

Principal Results of Pathologico-Anatomic Examination: 1. Complete obliteration of the lumen of the trunk of the portal vein over a length of 26.5 mm., extending from a point 23 mm. from the union of the splenic and superior mesenteric veins in the direction of the liver to the point of division of the portal vein in the right and left branches.

2. Cavernous tissue beginning at that part of the portal vein lying close to the liver and accompanying the intrahepatic branches of the portal vein for a short distance to the right and to the left.

3. Large dilatation of the cystic vein, of a vein in the ligamentum hepatoduodenale, indicated in figure 2 as *LC*, of the vessels of the omentum minus (hepatopetal vessels) and of the vessels of the stomach and duodenum (hepatofugal vessels).

4. A ruptured varix ventriculi as the cause of the severe hemorrhages.

5. An important increase in the size of the spleen together with an infarct of that organ.

6. The obliteration of the portal vein must be regarded as an organized thrombosis.

COMMENT

In our opinion, the most important clinical symptoms in this case can be explained fully by the results of the macroscopic and microscopic investigation. We found an abnormality such as existed in our case mentioned in literature, but not described in detail. Türk [9] thought that a clinical picture such as that in our case may arise as a consequence of a cavernous malformation in the terminal branches of the portal vein as the result of a thrombosis following an umbilical infection. But this idea, as far as we know, has not been confirmed by the results of a pathologico-anatomic investigation. Recently Naegeli [10] also mentioned the occurrence of a circumscribed cavernous formation of the ends of the portal vein after an umbilical infection. This condition could cause a typical clinical picture: infantilism combined with anemia and an enlarged spleen. Concerning this also we were unable to find a detailed description with pathologico-anatomic details.

There are also described in the literature a small number of cases of cavernous malformation of the portal vein,[11] but in every case the

9. Vorlesungen über klinische Hämatologie, 1912, p. 828.

10. Naegeli: Allgemeine Konstitutionslehre in naturwissenschaftlicher und medizinischer Betrachtung, Berlin, Julius Springer, 1927.

11. Klemperer, R.: Cavernomatous Transformation of Portal Vein, Arch. Path. **6**:353 (Sept.) 1928. De Jong, R. de J.: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **24**:160, 1911-1912. Hulk, J. F.: Nederl. Tijdschr. v. Geneesk. **1**:45, 1923.

stem of the portal vein was affected and not alone the part that lies nearest the liver. In those cases, also, the cavernous tissue was visible macroscopically in the ligamentum hepatoduodenale. Once only the cavernous malformation continued around the intrahepatic branches. In our case, however, the cavernous tissue was situated principally around the intrahepatic branches of the portal vein and only for a short distance around that part of the stem of the portal vein that lies close to the liver.

Further, our patient was 4 years of age, and, as far as we could find,[11] cavernous malformation of the portal vein has never before been observed in a patient so young. Most of these malformations were observed in patients between the ages of 25 and 55; Hart [12] described a case in a woman aged 20 years.

As this abnormality occurred in such a young patient, we first thought of a congenital abnormality, i. e., an abnormality of the portal system. This, however, was improbable, as may be concluded from the fact that all the roots and nearly all the intrahepatic branches of the portal vein had developed normally. It seems strange that only the trunk of the portal vein lying between the roots and the intrahepatic branches should have developed as cavernous tissue. This conception, accepted by Hart,[12] is also totally contradictory to the fact that in the serial sections we have been able to follow the whole course of the portal vein and its continuation into two branches, so that the anatomic make-up of the vein must have been fairly normal. We found, however, that the portal vein was small, as may be seen from the sections and our picture. Now it is known that the portal vein receives its real function value only after birth, when it also slowly enlarges. The fact that we found a rather small portal vein makes us believe that the closure of the portal vein must have occurred soon after birth.

What was the cause, then, of the occlusion of that part of the trunk of the portal vein adjacent to the liver? We are reminded of the idea originally expressed by Türk; viz., that following an umbilical infection a pylephlebitis may originally arise through the transmission of the infection of the umbilical vein via the left branch of the portal vein. It is true that in our case there probably was no question of a distinct umbilical infection. But a slight umbilical infection can exist without any special symptoms, or there may even be a harmless formation of granuloma. Indeed, we believe that the cause of the development of the malformation in our patient must be sought in this direction. If this conception is true, then the origin of the cavernous tissue around the part of the trunk of the portal vein adjoining the liver and around the intrahepatic branches must be explained in the following way: The

12. Hart: Berl. klin. Wchnschr., 1913, no. 48, p. 2231.

disturbance in the circulation of the portal vein was followed by the development of many small collateral veins around the portal vein, that is, the development of the cavernous tissue.

The facts that the liver was normally developed and that the total bodily structure of our patient was normal are contradictory to the idea of a congenital abnormality. It is true that Türk and Naegeli spoke of a cavernous malformation of the portal vein combined with infantilism, but, as has already been mentioned it is uncertain that in their cases a malformation like that in our case really existed. The fact mentioned by de Jong [13] that an analogous change of the portal vein has never been found congenitally is also against the theory of a congenital malformation in our case.

Yet the study of the relation of the fetal circulation to the liver during the different periods of growth shows that again and again vessels arise which later became impassable. *A priori* it is possible, therefore, that congenital malformation of the portal vein may occur. As far as the portal system is concerned, such cases are seldom found in the literature. It is unknown to us what the condition is in the not very rare cases in children who die shortly after birth, after having shown symptoms which pointed distinctly to a congenital malformation of the liver. It is a question whether the blood vessels of the liver have been examined sufficiently in these cases.

Another explanation for the cavernous tissue has been given by Versé,[14] Riesel [15] and Emmerich.[16] The authors believe that the cause must be sought in a thrombosis of the portal vein followed by organization and recanalization. This explanation does not hold true for our case, as the cavernous tissue was not found within the wall but around the portal vein, as it was found also in the case of cavernous malformation described by Hulk.[17]

Finally, Beitzke [18] and Hart [12] were of the opinion that they were dealing with a typical malformation, as they could find no traces of the wall of the portal vein in the cavernous tissue. In our case, however, we did find remnants of that wall.

In our case, these changes in the liver and spleen were very small. In regard to the liver, this is rather astonishing, because one expects great alterations in the liver in such an important malformation of the portal system. The size of the liver was normal, and microscopically no special observations were made. Also, the gallbladder and its contents appeared normal. In some analogous cases described in the litera-

13. De Jong (footnote 11, second reference).
14. Versé: Beitr. z. path. Anat. u. z. allg. Path. **48**:526, 1910.
15. Riesel: Deutsche med. Wchnschr. **35**:1685, 1909.
16. Emmerich: Frankf. Ztschr. f. Pathol. **10**:362, 1912.
17. Hulk (footnote 11, third reference).
18. Beitzke: Charité-Ann. **34**:406, 1910.

ture, small cirrhotic changes have been found, especially in that part of the liver surrounding the cavernous tissue. With these exceptions, the liver was nearly always normal. One must conclude that in these cases the liver obtained a sufficient supply of blood through the arteria hepatica, the cavernous tissue and the developed hepatopetal vessels.

The weight of the spleen was enormous, 1,070 Gm. There is much in favor of accepting the theory that the enlargement of the spleen was caused by the obstruction of the blood stream in the stem of the portal vein, i. e., by congestion. So in this case we should be dealing only with a congested spleen.

It is in agreement with this conception that we have sometimes observed, like many authors in analogous cases, that after severe hematemesis the spleen has greatly decreased in size. On macroscopic, as well as on microscopic, examination, it appeared, however, that the enlargement of the spleen in our case could not be explained by a chronic passive congestion alone. On microscopic examination, no marked deviations were found at the trabeculae and at the follicles. In the predominating pulp was found a rather large number of fibers; this was due to an increase in the fine connective tissue. All these observations are more in favor of hyperplasia than of chronic congestion. A fully satisfactory explanation of the enlargement of the spleen in such processes fails, as does that for the enlargement of the spleen in cirrhosis of the liver. In regard to the collateral circulation, we found on the surface of the body only a fine net of veins on both flanks.

As there was no correlation between the clinical diagnosis of chronic thrombosis of the splenic vein and the result of the autopsy in our case, we thought it useful, in relation to the therapy in this condition, to investigate how far such an agreement was found in the cases of chronic thrombosis of the splenic vein reported in the literature, especially in the cases that occurred in childhood.

In speaking of the morbid picture of chronic thrombosis of the splenic vein, one is accustomed to think of clinical pictures in which the process primarily or secondarily giving rise to a thrombosis of the splenic vein or of a part of the portal vein into which the splenic vein passes had already ceased. The morbid picture in such cases is dominated altogether by the thrombosis with its accompanying symptoms. The process giving rise to the thrombosis is usually an inflammation or a malignant tumor. This morbid picture is regarded as rare. The number of cases reported in the literature up to the present time in which the diagnosis of chronic thrombosis of the splenic vein had been made during life is important; however, only in a small number of these cases was the diagnosis confirmed at operation or at autopsy (compare the observations of Kretz [19] and Wallgren [1]).

19. Kretz, J.: Wien. Arch. f. inn. Med. **13**:249, 1927.

The differential diagnosis of this form of splenomegaly from other forms therefore often gives rise to great difficulties.[20] The number of cases, especially in childhood, in which the diagnosis of thrombosis of the splenic vein was confirmed at operation or at autopsy is very small. There are several indications that the etiology of this morbid picture in children often differs from that in adults, and that in children it often is as already mentioned, an umbilical infection. We believe that this fact makes it comprehensible why in children, though all the symptoms of the clinical picture of the chronic thrombosis of the splenic vein were present, splenectomy rarely showed thrombosis of the splenic vein.

After the recent and extensive publications on the subject by Wallgren and by Smith and Howard, and their survey of the literature, it is superfluous to discuss in detail all the cases reported in order to ascertain how often the diagnosis of chronic thrombosis of the splenic vein was confirmed at autopsy or by splenectomy. Autopsy could be performed in only a few cases reported as occurring during childhood. The cases mentioned as cases occurring in children have been reported by Finkelstein,[21] Wagner,[22] Westkott [23] and Hutchison.[24] The case of Hutchison deserves special attention here. A definite etiology was lacking in this case. A boy, aged 5, in the last two years before the case was reported had vomited blood several times. Ten days after his entrance into the hospital, he developed hematemesis and had a high fever. Two weeks after entrance to the hospital, he died. At autopsy the following conditions were found: "antemortem thrombosis of the splenic vein and also recent clotting in some of the mesenteric veins." The only data given concerning the liver were that this organ was large, pale, and not cirrhotic. Nothing is told about the stem of the portal vein.

We can say that of all the cases of chronic thrombosis of the splenic vein occurring during childhood reported in the literature, the case of Hutchison is perhaps the only one in which the diagnosis was fully confirmed at autopsy. In this case the clinical picture might be explained by a thrombosis of the splenic vein if the observations at autopsy may be understood in this way: "There was an old thrombosis of the splenic vein and a fresh thrombosis in some of the mesenteric veins."

Further, we can say that in children in whom splenectomy was performed, almost no change was found in the splenic vein, portal vein and liver, i. e., to the extent to which such changes may be found at

20. Morawitz: Klin. Wchnschr. **1**:731, 1928. Eppinger: Die Hepato-lienalen Erkrankungen, Berlin, 1920.

21. Finkelstein: Klin. Wchschr. **2**:1874, 1927.

22. Wagner: Klin. Wchnschr. **2**:381, 1928.

23. Westkott: Jahrb. f. Kinderh. **120**:232, 1928.

24. Hutchison, R.: Brit. M. J. **2**:281, 1928; ibid. p. 203.

operation. An exception may perhaps be made of the case reported by Bardach [25] and of one of the cases of Opitz.[26]

In agreement with the facts mentioned it is our opinion that the pathologic picture found in our case, which we think would not have been recognized at operation, occurs frequently. The etiology of cavernous malformation around the portal vein which we have accepted as being the most probable, in accordance with Türk, therefore cannot be excluded as frequently playing a rôle. In this respect we wish only to repeat as our opinion that for the occurrence of this pathologic picture there need not necessarily have been a severe umbilical infection in the history of the patient.

We shall also state some observations in relation to the clinical picture. In regard to the hemorrhages, it appears from the communications of several investigators that they often occur immediately after, or at the time of, a period of fever. Perhaps this occurred also in our case. How far one is justified in speaking of a relationship between the occurrence of hemorrhages and a rise in temperature can be known only after further thorough observation. There are several possible explanations for such a relation. Wagner,[22] who accepted a relation between hemorrhage and fever, gave the following explanation. As the fever is a symptom of an infectious process, it gives rise to an increase of the blood supply of the spleen, and this in turn increases the blood flow through the portal system, which facilitates a hemorrhage. There are, however, other explanations. The fever might also be a symptom of the formation of fresh thrombosis in the formed collateral vessels such as were also present in our preparation. Finally, the possibility exists that one has to do with a resorption fever caused by blood-destruction products. In the clinical picture of the chronic thrombosis of the splenic vein, the possibility of hemorrhages from other organs than from the stomach and intestine has been sufficiently discussed in the paper of Wallgren.

On repeated examinations of the blood of our patient, leukopenia was always found. Most investigators who reported cases which gave the impression clinically of being cases of chronic thrombosis of the splenic vein have described such leukopenia, which is not typical, since in western Europe it has been found in many forms of splenomegaly. Therefore, we cannot agree with Smith and Howard that a distinct leukopenia does not belong to the picture of chronic thrombosis of the splenic vein.

Most investigators who paid attention to this found that with chronic thrombosis of the splenic vein there was also a large decrease in the number of blood platelets.

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25. Bardach, M.: Arch. f. Kinderh. **71**:270, 1922.
26. Opitz, Hans: Jahrb. f. Kinderh. **107**:211, 1924.

We shall say a few words concerning the localization of the obstruction in the portal system during life, and without operation, i. e., exclusively on the evidence provided by the clinical symptoms, as was done by Opitz. When symptoms such as ascites, diarrhea and ectasia of the veins on the abdominal wall are present, one is accustomed to think of a stenosis in the portal vein. In our case, only the one symptom—fine ectatic veins on both flanks—was present, and we thought that we were not justified in ascribing any special significance for the localization of the process to this symptom. Ascites and diarrhea appeared only at the end of the disease, when the general condition of the patient became very poor. Consequently, we thought that the symptom of ascites and diarrhea pointed to the development of a thrombotic process from the splenic vein to the portal vein. However, autopsy showed that neither of these conditions existed, but that the place of obstruction in the circulation was situated principally in that part of the portal vein which lay close to the liver. Thus in our case the clinical symptoms did not give sufficient indications to determine in time the localization of the obstruction in the portal system.

Splenectomy is not generally recommended in a case with such a clinical picture. Several authors have observed that after the operation a new formation of the thrombosis with the accompanying dangers—further development of the process and emboli—occurred. After a severe hematemesis, it is of course necessary to improve the patient's condition by one or more transfusions of blood before the operation can be performed.

Kretz [19] cited some authors who lost the majority of their patients immediately after the splenectomy. Block [27] regarded it impossible to cure patients with chronic thrombosis of the splenic vein by splenectomy.

From our communication, it appears that one cannot predict where the cause of the obstruction in the circulation is situated. The presence or absence of ascites or the more or less developed ectatic veins on the abdominal wall has only small value in this respect. In adults, the stenosis seems to be situated mostly at the place where the splenic vein passes into the portal vein (Eppinger, Kretz), as has already been stated. The experience gained from cases in children that have been controlled by operation or autopsy is still too meager to give any certainty about this point; when splenectomy was performed, deviations in the splenic vein were rarely found.

As against the poor results obtained in adults by many authors, we can put forward the fairly good results obtained by Opitz, Wallgren, Smith and Howard in children. They were more successful in operations on patients who showed the clinical picture to the extent that

27. Block, W.: Deutsche med. Wchnschr. **53**:2112, 1927.

these children were still alive and in good condition for a long time after the splenectomy. Was it only by accident that these investigators obtained favorable results, whereas in adults the results were rather unfavorable? We could give several reasons to show why the operative treatment for chronic thrombosis of the splenic vein in adults gives less favorable results than in children. We mention the facts that the etiology of the clinical picture in adults often is a dangerous illness in itself, and further that the danger of emboli in adults is greater than in children.

We shall discuss briefly the question as to what extent one may expect a more permanent favorable result in those cases in which the place of the obstruction of the circulation in the portal system cannot be removed by operation, as in our case and probably in the majority of cases in children and in adults. In interpreting the effect of splenectomy, one must think of the important part the spleen plays in the circulation, as shown by the important investigations of Barcroft and his co-workers. Removal of the spleen thus means excluding from the circulation an organ with an important afflux of blood and therefore also with an important efflux of blood, and at the same time acting to a high degree as a reservoir for blood. Even when the place of obstruction in the circulation of the portal system is situated at a distance from the spleen, splenectomy is an important factor in ameliorating the congested circulation. Of course, we cannot predict whether or not the improvement caused by splenectomy will be permanent in such cases, since the cause of the obstruction has not been removed. Therefore, we cannot share the opinion of Smith and Howard that children with the clinical picture of chronic thrombosis of the splenic vein may be cured by splenectomy. In this respect, we prefer Wallgren's opinion:

Je n'ose pourtant pas fonder de trop brillants espoirs sur l'avenir de ces enfants. Je l'ai dit plus haut, après un certain temps on a vu reparaître chez des adults de nouvelles hémorrhagies ou survenir de la gangrène intestinale et cela en dépit d'une splénectomie antérieure. Les conditions étaient-elles différentes de celles des enfants opérés? Il est un peu difficile de répondre, mais ces faits nous commandent un pronostic réservé. And at another place: Aussi longtemps que les altérations persisteront dans des limites mesurées et ne provoqueront pas de thromboses des veines mesentériques, l'enfant a des chances d'être exempt d'accidents, mais si l'on peut s'exprimer ainsi, il marche sur un volcan; un jour ou l'autre de nouvelles thromboses peuvent survenir et entrainer une issue fatale. (Nevertheless I dare not make a too favorable prognosis for these children. As I have already said, after a certain time additional hemorrhages and gangrene of the intestine have occurred in some of the adults, and this in spite of a previous splenectomy. Were the conditions different from those in children who were operated on? It is somewhat difficult to answer this question, but in consideration of these facts one should be careful in making a prognosis.) And at another place: (As long as the changes are not marked and do not produce thrombosis of the mesenteric veins the child has chances of being exempt from accidents

but, if one may express himself thus, he is walking on a volcano; eventually new thromboses may occur and cause the death of the patient.)

Although splenectomy offers some advantages, one must always take into account the possibility of grave dangers of the operation. This makes a decision in favor of operating on a patient very difficult; each case should therefore be carefully examined and considered.

Apart from the difficulty of making a decision for splenectomy, the prognosis in chronic thrombosis of the splenic vein should always be made with the greatest prudence. The patients may remain in good condition for many years without an operation, but even after twenty years and more dangerous conditions may develop (Finkelstein, and Smith and Howard). Whether the patient could be cured without an operation is difficult to say, because of the great difficulty of making an exact diagnosis.

Roentgen treatment for chronic thrombosis of the splenic vein, sometimes recommended in the case of adults, need not be discussed here. Of this so-called therapy for this clinical entity there has been sufficient disapproval.

PRIMARY ANEMIA IN THE NEW-BORN *

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AND
EMMETT E. SAPPINGTON, M.D.
SAN FRANCISCO

While secondary anemia in the new-born is not an uncommon observation, primary anemia is extremely rare, according to reports in medical literature.

The first case was reported by Ecklin [1] in 1919. Blood examination made on the twelfth day showed: hemoglobin, 32 per cent; erythrocytes, 2,500,000, and leukocytes, 40,000. The family history in this case was important as a possible cause for the anemia, in that the father had heart disease, the mother suffered from grip and bronchitis while six months pregnant, and there was an indefinite history of the death of several infants (one of an infant 3 days old) before the birth of the patient. There was a normal blood picture at the age of 7 months.

In the second case, reported by Sustrunk [2] in 1924, the first blood count made on the third day showed marked anemia. The patient died at 10 days of age. Necropsy did not show any abnormality of the hemopoietic system but it did show valvular hematomas, hemorrhages of the renal pelvis and bladder and some icterus.

The third case was reported by Donnally [3] in 1924. The first count made on the twelfth day showed: hemoglobin, 20 per cent, and erythrocytes, 918,000. Two transfusions of citrated blood of 100 cc. and 70 cc. were given. The hemoglobin and the erythrocytes increased gradually, and the blood picture and other laboratory observations failed to show a cause for the anemia.

Stanford [4] reported the fourth case in 1924. In this patient, the blood was examined one hour after birth and showed: hemoglobin, 48 per cent; erythrocytes, 2,500,000, and leukocytes, 68,000. Without any treatment (except slight exposure to the roentgen rays in taking roent-

* Submitted for publication, Oct. 26, 1929.

1. Ecklin: Anemia of the New-Born, Monatschr. f. Kinderh. **15**:425, 1919.

2. Sustrunk: Severe Anemia in the New-Born, Ztschr. f. Kinderh. **38**:568 (Aug.) 1924.

3. Donnally, A. H.: Anemia in the New-Born, Am. J. Dis. Child. **27**:369 (April) 1924.

4. Stanford, N. H.: Anemia in a New-Born, Am. J. Dis. Child. **30**:19 (July) 1925.

genograms of the long bones), the hemoglobin and erythrocytes increased progressively, and on the twenty-third day the blood picture was within normal limits.

The fifth case was reported by Bonar [5] in 1925. (The mother had had seven previous pregnancies in rapid succession.) Blood counts made on the thirteenth day showed: hemoglobin, 31 per cent; erythrocytes, 1,200,000, and leukocytes, 18,000. On the twenty-first day, the hemoglobin fell to 23 per cent. Here again no treatment was given (except, perhaps, roentgen treatments when plates of the long bones were taken). When the patient was seen again at the age of 1 year, the blood picture was: hemoglobin, 99 per cent, and erythrocytes, 4,400,000.

In 1927, Canino [6] reported the sixth case. On the twelfth day, hemoglobin was 45 per cent; erythrocytes, 2,500,000, and leukocytes, 14,400. Except for a small umbilical granuloma and a slight mycotic stomatitis which healed rapidly, the family history, the results of the physical examinations and the laboratory observations were normal. No active treatment was given, and by the fourth month, the hemoglobin was 80 per cent and the erythrocytes, 4,500.000.

A seventh case was reported by Ehrmann [7] in 1929. The family history was unimportant. Blood examinations on the fifth day recorded: hemoglobin, 28 per cent; erythrocytes, 1,228,000, and leukocytes, 20,550. By the seventh day, the hemoglobin had dropped to 20 per cent. The laboratory reports, including bleeding and clotting time, Wassermann examination of the blood, fragility test, urine and stool tests, and the results of the roentgen examination of the long bones were normal. Two transfusions of citrated blood (one of 60 cc. on the seventh day and one of 100 cc. on the twelfth day) were given intraperitoneally. By the seventh month, the patient had apparently made a complete recovery.

COMMENT

Unexplained anemia in the new-born is not a new observation. In 1922, Holt and Howland [8] noted that anemia sometimes occurs in the first two or three months of life in a severe form without any discernible cause.

The normal new-born infant has a high hemoglobin and a high erythrocyte and leukocyte count. Lucas and Washburn,[9] in presenting the

5. Bonar, B. E.: Anemia in the New-Born, Am. J. Dis. Child. **33**:226 (Feb.) 1927.

6. Canino, R.: Anemia in the New-Born, Pediatria **35**:1200 (Dec.) 1927.

7. Ehrmann, E. W.: Anemia in the New-Born Infant, Am. J. Dis. Child. **37**:138 (Jan.) 1929.

8. Holt, L., and Howland, J.: Diseases of Infancy and Childhood, New York, D. Appleton & Company, 1922, p. 814.

possible variation in the blood counts of normal infants, noted that at birth the average healthy baby has an erythrocyte count of from 5,500,000 to 8,000,000. The hemoglobin varies from 95 per cent to 105 per cent (Sahli); the leukocyte count, from 14,000 to 25,000. By the sixth month, the counts show: erythrocytes, 4,000,000 to 5,000,000; hemoglobin, 75 per cent to 90 per cent, and leukocytes, 10,000 to 15,000.

Mackay,[10] in an investigation of nutritional anemia in infancy comprising 770 cases with 3,100 hemoglobin estimations, found that the hemoglobin in the new-born is high (about 110 per cent). Then there

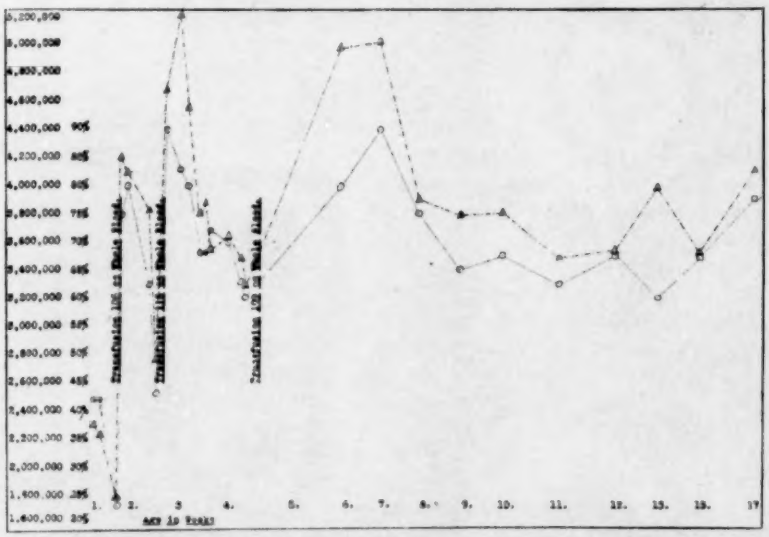

Chart 1.—Congenital anemia. Hemoglobin and red blood cell counts over four months. Circle and dotted line indicates hemoglobin; triangle and dot and dash line indicates red blood cells.

is a drop during the first few weeks of life to about 65 per cent at the age of 2 months. This is followed by a rise to 70 per cent at the age of 5 months.

Donnally [9] pointed out that the babies destined from antenatal causes to become anemic after the lapse of several weeks or months almost always at the time of birth manifest the hemoglobin content and the erythrocyte count of the normal healthy new-born infant. It is significant

9. Lucas, W. Palmer, and Washburn, Alfred H.: Diseases of the Blood and Blood-Building Organs (Haynes Clinical Pediatrics), New York, D. Appleton & Company, 1928, vol. 11, p. 39.

10. Mackay, Helen M. M.: Proc. Roy. Soc. Med. **22**:385 (Feb.) 1929.

that in all but three of the cases reported in the foregoing paragraphs severe anemia did not develop until about the tenth day.

Grulee,[11] in discussing congenital anemia, noted that it is only within the last few years that anemias of the newly born have been recognized; this, of course, does not mean anemias due to hemorrhage. He stated that these patients progress favorably as a rule, and although the anemia may be rather profound, it usually disappears without any special treatment by the time the infant is about 2 months of age.

In order to discover the factors operating to produce severe anemia in the new-born, we must look for evidence on the side of the mother or in the patient. Causes in the mother may be; acute illness while

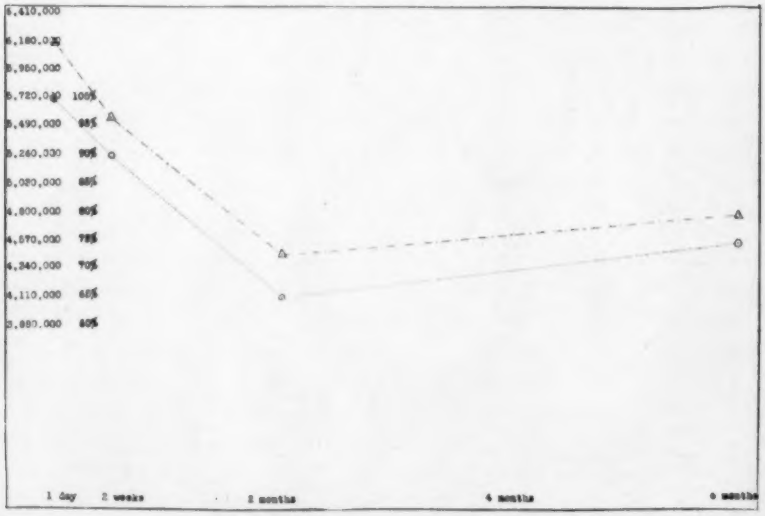

Chart 2.—Normal new-born infant. Hemoglobin and red blood cell counts from birth to six months. Dotted line indicates hemoglobin; triangle, dot and dash line indicates the red blood cells.

pregnant, tuberculosis, syphilis, mothers of advanced age or of weakened condition, repeated pregnancies at short intervals with abortions or hemorrhages or the use of hemolytic drugs.

On the side of the infant, such conditions as infections with pyogenic organisms, tuberculosis, active congenital syphilis, malaria, spontaneous hemorrhages, including hemorrhagic diseases of the new-born and hemolytic jaundice, may produce severe anemia.

Although in the first two and in the fifth cases cited, there may have been facts in the history or conditions of the infant to account for

11. Grulee, Clifford G.: Nebraska M. J. **14**:97 (March) 1929.

the anemia on the basis of factors known to produce anemia, the remaining five cases have apparently nothing to explain the etiology of the markedly reduced red blood cell counts and hemoglobin.

REPORT OF A CASE

The case here presented is that of a boy, born on May 2, 1929, after a full term pregnancy and normal delivery, whose weight at birth was 7 pounds 7 ounces (3.2 Kg.). The father, a physician, was young and in good health. The mother, whose general health had been good, had had an abortion at six weeks with good recovery in 1924. In 1926, a kink in the ureter caused considerable pain, but ended with good recovery and no recurrence. In 1927, she had an appendectomy with good recovery. The second pregnancy resulted in the birth of a boy, at term and in good condition.

The patient was the product of the third pregnancy. About ten days before term, a small abscess over the right labia developed in the mother. This was drained. After an uneventful delivery, several small abscesses developed on other parts of the body. The abscesses were drained and cultures from them were all sterile. The mother's Wassermann test was negative. Her blood counts were within normal limits. The urine was normal and she had practically no fever during the course of any of the abscesses.

At birth, the child cried spontaneously. He showed a slight generalized pallor. There was a small amount of puffiness about the eyelids. He had a soft systolic murmur, and the spleen was hardly palpable. He was breast fed for one week and then placed on a formula of milk, water and dextrose syrup. At the same time, he was given one-half ampule of liver extract daily. At the age of 3 months, the patient was put on a diet of lactic acid milk.

A blood count made on the seventh day of life, May 10, showed: hemoglobin, 42 per cent; erythrocytes, 2,300,000, and leukocytes, 12,400. The next day, the hemoglobin was 42 per cent, and erythrocytes, 2,230,000; the differential count was: polymorphoneutrophils, 56 per cent; eosinophils, 3 per cent; lymphocytes, 41 per cent. The blood smear showed very slight anisocytosis and poikilocytosis with slight polychromatophilia. The bleeding time was one minute; the coagulation time was six minutes. On May 13, the urine examination was normal.

On May 14, the blood picture had changed to: hemoglobin, 23 per cent; erythrocytes, 1,749,000; leukocytes, 20,500. The patient's blood was typed, and as the father's blood was compatible, the latter was used as a donor, and the patient was given 105 cc. of whole blood intravenously. On May 15, the blood count showed: hemoglobin, 75 per cent; erythrocytes, 4,220,000, and leukocytes, 22,500. A platelet count showed 749,555. On the following day, the blood picture was: hemoglobin, 80 per cent; erythrocytes, 4,100,000, and leukocytes, 11,400 (see chart of blood counts). On May 20, the blood count showed the hemoglobin to be 62 per cent; the erythrocytes, 3,820,000, and the leukocytes, 10,400. An examination of the smears showed the following: mature polymorphonuclears, 50 per cent; immature, 11 per cent; myelocytes, 5 per cent; eosinophils, 6 per cent; basophils, 0 per cent; large lymphocytes, 6 per cent; small lymphocytes, 20 per cent, and unclassified forms, 2 per cent and nucleated erythrocytes in ratio of 1 per hundred leukocytes.

The erythrocytes showed slight variations in the size with a tendency to macrocytes, and slight variations in staining with occasional polychromatophilia, but no achromia and no variation in shape. The general appearances of the smear did

not suggest such a severe anemia as the blood counts showed to be present. An analysis of the percentages of cells suggested that there was active regeneration of both the erythrocytes and the leukocytes of bone-marrow origin.[12]

On May 21, the hemoglobin had dropped to 43 per cent; the erythrocytes numbered 2,760,000. On the same day the child was given a second transfusion of 115 cc. of whole blood intravenously. Two days later, the count showed: a hemoglobin of 90 per cent, erythrocytes 4,680,000 and leukocytes 9,300.

With counts at intervals of practically every other day (see table) the hemoglobin and the erythrocyte count dropped consistently, and on June 6 showed the

Blood Counts in Case Presented

Date	Age	Hemoglobin, per Cent	Red Blood Cells	White Blood Cells
5/10/29	7 days	42	2,300,000	12,480
5/11/29	8 days	42	2,230,000	13,100
5/14/29	11 days	23	1,740,000	20,500
5/14/29	Transfusion of 105 cc. of whole blood intravenously			
5/15/29	12 days	75	4,200,000	22,500
5/16/29	13 days	80	4,100,000	11,450
5/20/29	17 days	62	3,820,000	10,400
5/21/29	18 days	43	2,760,000	8,400
5/21/29	Transfusion of 115 cc. of whole blood			
5/23/29	20 days	90	4,680,000	9,300
5/25/29	22 days	84	5,260,000	14,000
5/27/29	24 days	80	4,570,000	19,500
5/29/29	26 days	68	3,800,000	22,100
5/30/29	27 days	68	3,880,000	16,000
5/31/29	28 days	72	3,530,000	14,900
6/ 3/29	31 days	70	3,610,000	11,800
6/ 5/29	33 days	62	3,470,000	13,500
6/ 6/29	34 days	60	3,280,000	11,400
6/ 8/29	Transfusion of 100 cc. of whole blood			
6/18/29	46 days	80	4,990,000	10,400
6/25/29	53 days	90	5,010,000	10,600
7/ 2/29	8 weeks	75	3,900,000	8,500
7/ 9/29	9 weeks	65	3,790,000	7,000
7/16/29	10 weeks	68	3,800,000	8,100
7/23/29	11 weeks	62	3,480,000	9,000
7/30/29	12 weeks	68	3,520,000	7,700
8/ 6/29	13 weeks	60	3,980,000	9,800
8/27/29	15 weeks	68	3,500,000	6,500
9/10/29	17 weeks	78	4,110,000	6,700

hemoglobin, 60 per cent, and the erythrocytes, 3,280,000. A third transfusion of 100 cc. of whole blood was given intravenously on June 8. As shown by the charts, follow up blood counts have shown the hemoglobin to remain between 60 and 75 per cent and the erythrocytes about 3,810,000.

The last count, taken on September 10, when the baby was about 4 months of age, showed the hemoglobin content to be 78 per cent, and the erythrocytes, 4,110,000. The patient on that date had an excellent color, and the results of the physicial examination were entirely normal.

A blood examination of the mother, made on September 10, showed the following: hemoglobin, 80 per cent; erythrocytes, 4,250,000, and leukocytes, 6,000. The differential count was normal.

12. Washburn, A. W.: Personal communication.

SUMMARY

A case of congenital anemia is reported, being the eighth to appear in the literature. The patient, a boy, born after full term and in normal delivery, showed a marked primary anemia during the first week of life. His treatment consisted of three transfusions of whole blood intravenously of approximately 100 cc. each. He was also given liver extract daily.

As to etiology of the severe anemia in this case, it may be stated that the history of chronic abscesses in the mother may have been a factor. However, this is doubtful, since the mother's reaction to the infections, as shown by her blood counts and temperature, indicated no marked lowering of resistance. The contrast of the blood picture in this case with that of a normal new-born infant is shown by charts 1 and 2. Changes in the blood counts as a result of the three transfusions are also shown.

Congenital or primary anemia in the new-born apparently represents some cause, active during the prenatal period, which has markedly inhibited the production of red blood corpuscles and the normal amount of hemoglobin. The underlying cause of this condition is at present unexplained.

CORRECTION

In the article by Dr. B. J. Malnekoff, entitled, "Acute Mediastinal Abscess," which appeared in the March issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN (**39**:591, 1930), the legend for figure 1 should read: "A dense shadow is visible well inside of the anterior mediastinum and outside of the lung (stereoscopic)."

Progress in Pediatrics

WHY PARENTS CONSULT THE PEDIATRICIAN *

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One is constantly being reminded that some of the values of the family physician are being lost in the coldness of present day science and specialization. Of more recent years, the intangible personal services that a physician can give his patient have been the subject of discussion which is increasing as the tangible things, that is, scientific achievements, gain greater prominence and monopolize the physician's attention. Most of what is written is inspirational; it points out the problem and appeals for a solution, but does not contribute seriously to that solution. How can medical practice be carried on so that the patient as well as his organs can be recognized as behaving, causing disorder and responding to treatment? Some critics would even give up being so scientific in order to preserve some of the human contact with the patient. That solution, of course, is exactly what the cultist thrives on. Since physicians cannot, without quackery, cease to be scientific, why not put some of these intangible values of the family physician on a scientific basis so that they will not be lost from generation to generation and so that those without the knack can come to apply them? George W. Henry [1] has done this in the case of 300 patients seen in a hospital service. Last year I [2] made a study of patients with gastro-intestinal diseases to reveal the personal and social problems of these people, problems which bear on the production of the complaint and the treatment for the disorder. In the present study, made in a pediatric clinic, I am interested in the parent who brings the child to the physician. Study of that parent should be of definite practical value both in diagnosis and in treatment. Pediatricians are being bombarded constantly with instances of failure due to peculiarities in the personality of the parent. An objectification of these obstacles should augment whatever art has already been acquired in this regard, and

* Submitted for publication, Jan. 21, 1930.

1. Henry, George W.: M. J. & Rec. **130**:383 (Oct.) 1929.

2. Stevenson, George S.: Why Patients Consult the Gastro-Enterologist: Motive and Attitude of One Hundred and Fifty Patients, J. A. M. A. **94**:333 (Feb. 1) 1930.

make this part of the art of medicine available in usable form to the medical student whose experience has not yet provided him with a "feel" or an intuition about the parent of his pediatric patient. Apart from its value in treating a child for any special sickness, the peculiarities in the personality of the parent should be known to the physician who is called on to advise the family in maintaing the health of the child as a thing apart from a specific complaint. In other words, as advisor in child guidance he must know the emotional trends of the parent who is to help guide the child.

METHOD

In studying the parent for the purpose of objectifying these more personal technics and opportunities, I clarified the aims of the project in the beginning by stating two definite questions: Why does the parent bring the child to the physician? The answer to this, if it is to have value, cannot be merely a repetition or a classification of the parent's explanation of his purpose or the complaint, but must be a revealing of the parent's basic [3] motive or, as it turns out, the mixed motives that drive him to the physician. In short, what impelled the mother to seek medical aid? Often these motives agree with those assumed by the parent in stating the complaint; i. e., she brings the child as the fulfilment of parental responsibility, for example, in case 27. The mother is somewhat, though not unduly, concerned about her child's persistent cough. The diagnosis is pertussis. Often, however, the parent is unconscious of the real motive which has driven her to the physician; for example, in case 13 an aunt was using her nephew's illness to pry her brother and sister-in-law apart. She sought a diagnosis to prove that her sister-in-law had not taken good care of the child. It is obviously impossible to meet a parent on common ground and to influence him permanently if the motive is not understood. Furthermore, powerful forces are at times bound up in this motive which may be called on by the physician to strengthen his plan of treatment; e. g., in case 24 a man referred his nephew to a physician out of loyalty to his dead sister, the boy's mother. The second question is: How is the parent apt to balk the plan? What emotional facts must be considered in planning treatment? Often the ulterior motive, besides driving the

3. Basic is, of course, a relative term. What would be basic to the parent's complaint might be quite superficial in the parent's individual psychic make-up, and the basic motive in the psychic organism might be equally superficial physiologically. I consider here as basic the satisfaction which the parent derives from the process. To go beyond this point as a routine would be to set up a goal that would be blocked in treatment, if not during examination, because of limitations of time and technic.

mother to the physician, will likewise interfere with treatment. But many parents, sincere and conscious of their motives in bringing their children, and clear in the statement of a complaint, have problems of personality which have had little or nothing to do with their coming to the physician. These problems, nevertheless, may become involved in the plan of treatment that the physician prescribes for the child, perhaps because this plan strikes a note on which the mother is sensitive or because it demands things of her that she is not capable of doing; the child consequently suffers. Many of the problems of mismanagement, such as feeding problems, are of this type; e. g., in case 21 the mother follows the physician's orders, but with such poor spirit and obvious fear that the child will die in the process that the value is lost. The treatment may require a change in the routine, point of view or habit of a parent, and this test the parent frequently fails to meet. The result is that treatment fails, and the next physician is told that the first one did not understand what was wrong with the child. Sometimes these obstacles can be first detected and understood only as they arise in treatment. At times, however, it may be decided in advance during the initial examination that the personality of the parent will have to be given definite consideration in laying out a plan of treatment.

For the purposes of this study the parent was seen but once; immediately on coming to the physician and before any work had been done on the case. It consequently does not show the value that would come of studying the factors responsible for small failure of cooperation during treatment. Neither does it show the causes of larger failures which result in the parents giving up the plan of treatment completely. The mother who withdraws her child from treatment is as a rule so defensive or antagonistic that she offers obvious difficulties to a study designed to determine the reasons for the breakdown of cooperation. To a certain extent the present study touches on this point, in that in many cases I was the next physician and the parent expressed himself about my predecessor. This incomplete follow-up could not compare, however, with a study in which the dissatisfied parent could be seen by the person who had been aware of the full facts of the previous program of treatment. The real cause of the parent's dissatisfaction could be revealed and the physician would less frequently be made the "goat" of the parent's warped motives and hyperesthetic emotional complexes.

In order to answer these questions as to the parent's motive and probable cooperation, arrangements were made with the pediatric department of Cornell Clinic to interview the parent as soon as the child was admitted. There was no selection of cases. The parent was seen before any clinical examination had been made or any history taken. As far as possible, the parent's statements were recorded verbatim; at times

the parent's attitude toward the child as observed at the clinic was recorded. Later the facts of the pediatric examination and treatment were recorded also. No attempt was made to influence the pediatrician's study of the case. My study was a probing and in no wise an attempt to apply the data obtained. The therapeutic application is the next step.

It is more difficult to approach the personal problems of a parent than those of a patient with gastro-intestinal disease. The latter presents an immediate approach because he complains about himself, and to discuss the patient himself is quite pertinent. The parent, however, is farther removed from the complaint. No definite procedure can be given to establish the rapport which will permit a discussion of a mother when she comes to have her child examined or to lead the examination from the child to her. If the personal problems are approached too precipitously, the parent balks and feels that the child's problem is being lost sight of.

Probably the safest first step [4] in the interview is to allow the parent free rein in discussing the child's problem, even encouraging him to amplify this as fully as possible. It is essential from every standpoint that the complaint be clear. If a clear statement cannot be obtained, one should suspect that the problem lies in the parent rather than in the child. It is well to condense and reformulate the complaint in order to be sure of agreement, and to have clarified any vague terms used by the parent, such as "run-down" or "sickly." As the parent exhausts his thoughts about the child he will be more apt to discuss himself or to present an approach for such a discussion. Furthermore, having given the child full consideration, he is less sensitive to a discussion of his own personal problems and attitude.

The next step is to discover just what this illness means to the parent. Has it worried the mother severely? Is anxiety dominant in her attitude toward the problem? Is the anxiety based on adequate objective fact or is it a general concern about health out of proportion to the problem? Has this particular type of complaint or the problems surrounding it any connection with the parent's past experiences, particularly with calamities? Has any one close to the parent died or suffered from a similar complaint? In case 7 a woman complains that her child's knees hurt. She becomes panicky because her husband had to have both legs amputated for gangrene and she fears the same fate for her child. Such things as family susceptibility, similar illness among or known to acquaintances, superstitions and traditions about certain facts and their meaning come to evidence here; e. g., to many parents a large

4. It was, of course, necessary in this study to indicate to the parent in the beginning that another physician would continue the examination and prescribe treatment.

appetite means a tapeworm. Has the parent fixed ideas of what should constitute treatment? It is of importance in this connection to obtain the parent's idea of what caused the trouble; personal resentments having a close bearing on the child are thereby revealed. "My husband made me work when I was pregnant" is a powerful weapon in the hands of a deserted mother and soothing to her ego, especially if a sick child is at hand to clinch the argument. Has the illness caused the mother much inconvenience or annoyance? Does she fret about this?

If not obvious, it may be desirable to know how a parent happens to come for help at this place and at this time. Among other values, this may show the parent acting only under outside pressure and consequently apt to be less hearty in cooperation. It may reveal a more complex situation which has determined why the child has been brought at this time or to this place, a thing which must be known if an adequate program of treatment is to be outlined. Why in this long standing case did the parent not come for help before? Is she ignorant, or did not the child bother her enough to stir her up? Does she come now because somebody urges her to? If she did go to a physician earlier, why did he not satisfy her? What difficulties did she experience in previous treatment? Can they be overcome now that they are known? What is the family history of this parent; are there other children; what is their relationship to the parents? What is the relationship of the parents to each other?

It is, of course, impossible to follow out all the directions into which such an interview may lead the physician; the foregoing are merely general indications. The doctor must develop a sensitivity to leads and a technic for pursuing them. But this sensitivity can be developed only by paying routine attention to the more personal phases of a case. It is not sufficient to find merely something wrong with the parent or the situation in which he is living. To be of value for a coordinate plan of treatment for the parent and child, that "something wrong" must have a relationship to the child's problem. This does not of course preclude the separate handling of unrelated problems. This should be done and is greatly to the parent's personal benefit and to the child's potential welfare.

There are certain outstanding limitations to this study and the method used. The number of cases, thirty-two in all, is necessarily small. The study is of value in showing that certain facts exist in relation to the children and their parents. The study does not show, except broadly, how frequently such facts occur, nor does it assure a complete listing of these facts. It is quite certain that by studying a larger group, other mechanisms of behavior and other interesting and fairly frequently recurring social situations would be revealed.

RESULTS

Previous experience with cases of gastro-intestinal disease was sufficient to show that it would be impossible to ask all the questions in the beginning that would be answered in the end. In other words, answers would be found to questions that had not been thought of. The observations listed show that the same is true of this study.

An attempt has been made to classify the problems as they come to the pediatrician. Accordingly, there are:

Practically uncomplicated medical problems	12
Medical problems so exaggerated by the involved adult personalities that these personalities must be considered in any adequate plan of treatment	15
Practically entirely problems of the parent's or referrer's personality	5

The analysis of each case resulted in a decision as to the chief motive of the parent in referring the child. Motives of parents appeared as:

Altruistic (love)	24
Ulterior or hidden	8

The altruistic parent brings her child because common sense indicates that the condition of the child requires attention. These parents usually have in addition a certain amount of anxiety which is undoubtedly a driving force. In eleven cases this was estimated as somewhat excessive, in sixteen it was appropriate, and in five it was low; in only one case was the parent coming for relief from her own anxiety rather than to secure a healthy outlook for the child. Such excessive anxiety one must expect to find based on the past experiences of the parent more than on the observable conditions of the child, and a plan of treatment which ignores this parent's personality is far from adequate. In most cases the excessive anxiety is relieved by a clear explanation of the problem to the doctor.

Besides this one case of anxiety, the hidden motives of parents included:

Compensation for and a feeling of guilt for past indiscretions supposedly affecting the child; e. g., in case 1 the mother induced an abortion shortly before the conception of the patient. A serious infection resulted from this abortion. She felt that she gave the child a poor start in life. Consequently she sought atonement in overprotecting the child and tried to get relief from guilt in seeking medical opinion, which would negate or counteract her supposed misdeed.

Relief from annoyance caused by the child's trouble; e. g., in case 6, the mother said "I hope we don't have to go through this every year."

To establish a stronger case of neglect against a deserting husband by having a physician say the child is sick.

To expose the neglect of the child's mother in caring for the child by getting a diagnosis. This child's aunt who brought her for examination felt that the child's mother took away her brother's affection.

To avenge a dead sister by exposing her husband's neglect. This neglect was to be established by securing a diagnosis.

Unless one is aware that the child is being made the instrument of a hidden motive, much time and thought in laying out a plan of treatment may be in vain. One might well fear in two or three of these cases that unless the relative were weaned away from the original motive, any endeavor to put the child on a healthy basis would receive half-hearted support, if not actual opposition, unconscious though it might be.

Even when the motive may in the beginning have been altruistic or only secondarily fortified by personal factors, the parent may have a limited vision which may obstruct treatment if not taken into consideration. Chief among these parental myopias is the attitude of overprotection. The physician may achieve success in the individual illness without taking the parent's attitude into consideration because he has effective measures of combating the immediate problem or of reassuring the parent that it does not constitute a serious problem. But unless he deals with the overprotective attitude itself he is apt to find it recurring again and again in other forms of complaint, each requiring special examination and each undermining the morale of the child and cramping his growth in personality. A pediatrician once said to me: "My hardest job is to convince mothers that when a child has recovered from an acute illness, he is no longer sick."

An overprotective attitude on the part of parents was found to be caused by a variety of things:

The parent has committed errors in the past which he thinks may have affected the child and he is making up for this.

The child is or has been treated harshly or neglected by others, and the parent is trying to make up for this.

The child is achieving a parent's unattained ideal, e. g., in school, and the parent is guarding this vicarious success.

The child was previously sick and the parent continued to nurse it.

The parent knows of another case of illness, perhaps his own past illness, which he is afraid the child will suffer.

The child is teased by other children, so the parent tries to make up for this.

The parent is mentally weak and suggestible and gives in to the child easily.

Circumstances restrict the mother's attention to this one child, e. g., an only child or a foster child.

Besides overprotection, which limits the vision of a parent and so obstructs treatment, there are other often obstructing problems:

The parent does not understand a physician's contributions or functions.

The parent will not be bothered with the details of treatment.

The parent is inconsistent in her attitude toward the child, now anxious, now impatient.

The parent rejects the plan of treatment because it implies that she has committed errors in her job as a mother.
The parent is guided by superstitions and erroneous ideas.
The parent is worn out and discouraged by the fatigue and loss of sleep caused by the problem, and is low in morale.
The child's problem is dependent on a social situation which the parent is unable to alter.
The parent is misguided by the ulterior or unconscious motives referred to.
The parent is sick, overemotional or mentally incapacitated for cooperation in treatment.

It has been previously indicated that in the interviewing of the parent a fact of value is obtained if the reason for the failure of previous physicians is made clear. This shows what the parent was after, but did not get, and points out to the present physician the direction in which he will have to work to satisfy this parent, or the modification he will have to make in this parent's expectations of the physician, if these expectations are to be scientifically and ethically realized. Eighteen of the thirty-two parents expressed dissatisfaction; six were satisfied, and in eight this information was not obtained. The following explanations of dissatisfaction are valuable in this regard:

The physician said nothing was wrong.
The physician diagnosed but did not treat.
The physician failed to diagnose.
The physician varied diagnoses; he did not understand the case.
The physician did not attend to the complaint but found some other problem.
Treatment was unsuccessful.
The physician's motive was considered pecuniary.
The physician's examination was incomplete.
The physician said that the child would outgrow the trouble.

While some of these criticisms may be justified, the fact with which I am concerned is not criticism of medical practice, but that these represent attitudes of parents and as such, whether justified or not, cannot be ignored.

Diagnosis plays an important part in the relation between physician and patient or parent. It is significant of the breadth of perspective of this particular pediatric clinic, that next in frequency to diagnoses of disease of the tonsils and adenoids one finds the diagnosis, "parental mismanagement." In discussing the reasons why some patients found their physicians unsatisfactory, I have stated that at times the diagnosis did not meet or answer the complaint. Adolf Meyer [5] in discussing the complaint in connection with psychiatric cases said: "I like to keep in

5. Meyer, Adolf: The "Complaint" as the Center of Genetic-Dynamic and Nosological Teaching in Psychiatry, New England J. Med. **199**:360 (Aug. 23) 1928.

evidence the question of complaints, of the family, of the patient, those who are acutely involved and not inclined to be satisfied with mere routine. . . . In the end the task is the amplification and specification of the complaint expressed in the most factual, objective and potent terms. . . ." When a mother complains that her child has a vaginal discharge one cannot without detailed explanation expect to hold her attention and cooperation with a diagnosis of bronchitis and nasal catarrh. Neither will knock-knee, fallen arches and indigestion explain to a parent without much elaboration why a child sweats excessively. "Weak feet and tonsillitis" do not satisfy a parent who wants to know why her child vomited a worm. A mother who has difficulty getting her child to eat would want to know the connection between this and the diagnosis of chronic tonsillitis.

Similarly there are certain tautologic diagnoses which fail to satisfy the parent. Some of these diagnoses, because of their Latin names, render the parent mute so that he will not show his ignorance or render him impatient because he is left in the same darkness with which he came. Here are some that have been gleaned:

Complaint	Diagnosis
Head pain	Hyperesthesia
Overweight	Overweight
Pain in the knees	Arthralgia
Itching	Pruritus
Five pounds underweight	Malnutrition

Such diagnostic terms as indigestion, endocrine dysfunction (unspecified), constitutional inferiority and hyperactivity, while not exactly tautologic, are about as useless in planning treatment.

CONCLUSION

There are certain hidden motives whereby parents bring their children to the physician. These motives should not be ignored but should be incorporated in the diagnosis since they influence the cooperation of the parent in treatment. More attention should be paid to the broadening of medical diagnosis and therapy as set forth by Lewellys F. Barker as follows: "It is clear that medical diagnosis dare no longer be content with physical and chemical studies alone but must include also psychological and sociological studies of these persons." [6]

The parent has in addition other emotional and situational problems which will influence cooperation and which must be considered in planning effective therapy.

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6. Barker, Lewellys F.: The Growing Influence of Biological and Socio-Psychological Conceptions of Man upon Medical Research and Medical Practice, Lewellys F. Barker, Pan-American Medical Congress, Havana, Dec. 31, 1928.

These problems can be revealed by a study of the parent as a part of the study of the child according to the method described.

The form of presentation of the diagnosis is an important factor in cooperation.

The attitude of parental overprotection is the most serious obstacle to effective cooperation. This attitude can be analyzed into components intelligible to the parent, components themselves subject to therapeutic attack.

The following brief statement of each case sets forth the essential psychologic problems that must be recognized.

REPORT OF CASES

CASE 1.—The mother became pregnant, induced an abortion and suffered an almost fatal infection. Three months later she became pregnant again and experienced a normal course and parturition. However, she felt that her abortion gave the infant a poor uterine background; she blamed herself for every little indisposition that befell him and she watched him like a hawk. She complained of the child's sweating at night, restlessness, talkativeness, poor appetite and pain in the stomach and forehead. She said, "I was afraid he would get chorea. My sister-in-law knows a girl who got chorea. She is so curious it makes me upset."

CASE 2.—The father's anxiety about the child was excessive, as shown by his going from physician to physician and refusing to accept and act on the advice that the boy's headaches were not serious and that his eyes should be examined. There was probably a deeper cause for this anxiety, antedating the child, but it found legitimate expression here because the child had a disfiguring birthmark and was teased about it. The father was consequently overprotective. The mother also blamed herself and overcompensated in protecting the child because in her mind she was the cause of his birthmark. While pregnant she caused a puff of flame on her gas stove by delay in igniting the gas.

CASE 3.—The previous physician diagnosed the condition as albuminuria but prescribed no treatment. The mother wanted something done. The child had a cracking in her knees and was overweight.

CASE 4.—The mother was separated from her husband and felt vindictive. She had to support herself while pregnant and since then. She seized on every misfortune to herself and the child as another count against her husband and derived a certain satisfaction from the piling up of counts. Since she and the child were sick frequently she felt that they had both suffered through the father. She did not, however, worry unduly about the child. The child had had pneumonia twice and had always been under the care of a physician. In this instance, she had been having "indigestion" (gas) for a year and recently some fever (103 F.) and loss of weight (7 pounds [3.1 Kg.]). Her physician said it was her kidneys and then her liver. The mother lost confidence in him and so changed.

CASE 5.—The mother was overprotective of her child because the father was irritable with it and she had to make up for that; because the child had had a very severe attack of pertussis and in her mind needed unusual care, and because she had a craving for perfection which she satisfied by urging the child to the top of the class in school. The child complained of a ringing in her ears when

she met difficulties in school. She never developed this when at play, but she did seem to get it when the situation was indifferent. This suggested a real ear condition which had laid the foundation for a means of escape from trouble. Since previous physicians had not paid much attention to this complaint, the mother left them.

CASE 6.—The mother was at the same time selfish and worried about her child. She intermittently scolded and spoiled him. He had attacks of bronchitis and asthma. The child refused to be separated from the mother during the interview and lay on the floor and kicked; the mother slapped him in the face and said, "Come on, bad boy. I don't love you any more." She regretted his illness as much because it burdened her as because it threatened him. She said, "I hope we (she and the father) don't have to go through this every year."

CASE 7.—The mother complains that her child had pains in his knees and joints. She was not overly anxious but was sensitized by the fact that the father of the child had his legs amputated for gangrene. The previous physician did not relieve the condition.

CASE 8.—The mother had an overactive child[7] who said that his stomach hurt. He got up in the morning with puffy eyes, he had become thin, and was cranky and wild. The mother said: "His nerves seemed to be working all the time." He was constipated. She thought she caused this by feeding him too much when he was younger. She blamed his nervousness on the father's strictness, and she suspected that his behavior might be a way of getting attention.

CASE 9.—The mother was bossed by the child. In school the child was less powerful and missed the satisfaction of dominating others; he became nervous, restless and fidgety. The physician advised four weeks' rest in bed at home for chorea. The restlessness cleared up. Return to school precipitated the nervousness. The child was timid.

CASE 10.—The mother was concerned about the child's lack of appetite, vomiting and slight fever (102 F.). The physician said that it was habitual vomiting and that the child inherited nervousness from the mother. He also said that the trouble was caused by poor teeth. The mother was afraid that he did not understand the problem.

CASE 11.—Two years ago this boy was bitten by a monkey and shortly after began to have convulsions. These occurred particularly if his mother opposed him.

CASE 12.—This father and mother were worn out because of the severe eczema of their child, aged 4 months. They were appropriately anxious about her condition.

CASE 13.—An aunt was antagonistic to and jealous of her brother's wife, the child's mother. She accused her of neglecting the child. The child was delicate, constipated, ate poorly, had rings around her eyes and had vomited worms the previous summer.

CASE 14.—The child had painful urination and a discharge. "She has a cold in her bladder, has always been sick in her bladder."

CASE 15.—The child itched, particularly at night. Five or six physicians had been unable to help her.

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7. Overactive child is an empty term which tells no more than was already known. In its place should be an etiologic term explaining the cause of overactivity, such as fatigue, lack of sleep, chronic infection, chronic emotional pressure, undernutrition and intoxication.

CASE 16.—The father was worried because the child has only one descended testis. Friends had advised him to do something about it. He had been advised by his physician to have an operation for it, but he wanted to check before going ahead. The father had had considerable sickness and was sensitized to the need for preventive measures on this account.

CASE 17.—The grandmother was a member of a doting family in which the patient was the only child for six years. They were all worried about his headaches, the fact that he was 5 pounds (2.2 Kg.) underweight and his excitability, but had not had him under one physician continuously. He did not like to play with boys and they realized that he was spoiled to a degree; to them, however, that fact was a thing apart from the complaints.

CASE 18.—The mother and also the father had asthma, an experience which exaggerated their reaction to the recurring bronchitis of the patient. The boy was thin, nervous, bad tempered, talkative, restless and impulsive. He had had no regular physician.

CASE 19.—The mother had good cause for anxiety but seemed to control herself sufficiently to avoid spoiling the child. The boy had asthma and a draining mastoid. Physicians had been unsuccessful in treating him for these conditions.

CASE 20.—The foster-mother was quite objective about the serious awkwardness that had developed in this little girl's legs. She fell and could rise only by working her hands up her legs.

CASE 21.—The mother had trouble in getting her boy to eat. The family followed the physician's advice to let him starve but with such fear and trembling that the boy was quite aware of the hold that he had. The mother said: "He drives me crazy trying to get him to eat. I coax, I pay him, but he won't eat. My doctor does not help." The fact that the boy had severe bronchitis at 2 years of age gave the mother a poor start, and the father's feeling that the boy will die is no moral help to her.

CASE 22.—The mother was a weak person, who yielded to the boy. She bought him all sorts of things, such as a violin, in her effort to overcome his delinquency, but she gave him no guidance. Because of his minor delinquencies, she was about to send him away to a school. She wanted to know first "if his head was all right"; if he was "just bad" or perhaps "nervous or crazy." She was ignorant and she came from a district where delinquency was prevalent.

CASE 23.—The mother, who was foreign, was extremely suspicious of physicians; she thought that private physicians held patients unnecessarily long in order to make money. She was overprotective of the child because it came late —when she was 40 years of age, and twelve years after the preceding child. She had been told that the child had a glandular disorder (endocrine), but since another child of hers had cervical adenitis, she complained of the patient's neck rather than of the Froelich symptoms which were evident.

CASE 24.—A young uncle, in his solicitude for the patient, was expressing love for his dead sister and displacing the child's father whom he hated. The child had suffered considerably; he had rickets when he was younger, and now showed convulsive twitching, restlessness at night, crying spells and emotional instability. The uncle sat beside him at night to soothe him. There was probably a slight guilt factor in that the trouble came on while the family was away on a vacation and the boy was left with a substitute.

CASE 25.—The mother had but one child and she hoped it would be the last. She said, "I suffered too much with her. She is bad; not even one night she isn't

up." The child had a rash and did not sleep well. The physician whom they had gave no relief. The selfishness of this mother was a threat to a plan of treatment.

CASE 26.—The father had been told by his present doctor that a previous diagnosis of hermaphroditism was wrong, and he had been sent for a confirmatory opinion. The boy had one undescended testis. The father had been worried but not unduly so. He had seen the potential threat to the personality development of the boy in this physical abnormality (hypospadias) and had been careful about showing his concern. To date, the boy's emotional development had been good.

CASE 27.—The mother was somewhat, but not unduly, concerned about a cough which the child had.

CASE 28.—The mother was rather tense and concerned about her son, aged 11 years, who was restless, tense, easily embarrassed, blushed readily, was jerky and clumsy with his hands and sucked his thumb. His condition had been discussed so frequently that he is well aware of his abnormality. The mother was unwilling to take her physician's advice that the patient would outgrow this trouble. There were probably home influences that were not revealed by the interview. This case was of the type to which the tautologic term hyperactive child is often applied.

CASE 29.—The mother saw in the behavior problems of the child a matter for medical advice. The child recently had slept poorly, complained of her eyes aching and had eaten poorly. She became frightened at night and went to her parents' room, where she was allowed to remain until she fell asleep. The mother thought that the patient had heard the parents discuss terrorizing things which they read in the newspapers.

CASE 30.—The mother, who was emotional, was concerned about a recent increase in her child's weight. The child was sensitive to teasing; she sometimes fought and sometimes cried. This obesity constituted a definite psychic burden. The parents diverged in their training methods. The mother was strict when she reacted but usually reacted only when annoyed. The father was too lenient.

CASE 31.—The mother was appropriately worried because the child looked pale, had attacks of sore throat and frequently had a slight fever (100 F.). The patient lacked energy, was listless, ate poorly and was underweight. The child complained of headaches, was emotional and spent most of her time reading. Physicians had previously found nothing wrong, but they had never given her a complete examination.

CASE 32.—The mother was not satisfied with the physician's statement that the child would grow out of her diarrhea which had existed for four years. She said, "I thought they should have analyzed her stool." Now that the girl was starting school the mother saw the diarrhea as a more serious inconvenience, but was as well concerned with it as a matter of the child's health.

Pediatric Biographies

WILLIAM CADOGAN

1711–1797

JOHN RUHRÄH, M.D.
BALTIMORE

A good biography should have a setting consisting of an account of the time in which the subject of it lived and worked. In such thumbnail sketches this is not possible, but the following paragraph from "Pediatrics of the Past"[1] may help place Cadogan, one of the lustiest of the eighteenth century pediatrists:

> To truly estimate Cadogan, and he looms large, one must bear in mind that he graced a period, when, as a rule, "the mind like the beard had a formal cut." The eighteenth century was a formal age. Men bowed and scraped punctiliously over their snuff boxes; the philosophers droned tediously; even the music of the period was precise. Small wonder that the medicine of the century followed suit. The medical London of those days was replete with interest; the mere mention of a few names suffices to give it coloring. At that time John Brown was flourishing. A protégé of Culen, he announced a theory known as the Brunonian, a theory actually taken seriously for a quarter of a century and which comes down to us even until today in the textbook use of the words *sthenic* and *asthenic*. Smellie was doing obstetrics, the Hunters and Percival Pott, surgery, and medicine was represented by such men as Cullen, the older Heberden and John Fothergill, when Cadogan decided to give up his work in Bristol and move up to London to make a place and a name for himself.

Only the barest outline of his life is known. He was born in 1711, and studied at Oxford, from whence he was graduated with the degree of B.A. in 1731. He then traveled on the continent to study medicine, and received his doctor's degree at Leyden in 1737. He entered the army and later settled in Bristol, where he was living in 1752, when he was made a Fellow of the Royal Society. Shortly after he removed to London, where in 1754 he was made physician to the Foundling Hospital. The following June he received his M.A., M.B. and M.D. from Oxford, and in 1758, he was elected to membership in the College of Physicians. He served four times as censor, and in 1764 and 1792, he delivered the Harveian oration. His portrait by R. E. Pine hangs on the walls of the College. In London, he lived in George's Street, Cavendish Square.

He did not write much, but when he did he said what he had to say in a clear forcible manner, which was not considered quite polite

in those days when a lack of enthusiasm for everything was regarded as the chief requisite of culture. His "Essay on Gout," published in 1771, created a storm. It ran through ten editions in two years and was the talk of the town. He advised diet, simple living and exercise! It is worth reading, as is the poem about it published anonymously by a Mrs. Ireland. Both will be found in "William Cadogan, His Essay on Gout," published by Hoeber in 1925.

His chief pediatric contribution is "An Essay Upon the Nursing and the Management of Children," published in London in 1750. Nine editions appeared in the next twenty years. The smugness of English pediatrics received a severe shock from this just as had the complacent physicians who treated their gouty patients with drugs instead of diet. Cadogan had clearcut ideas of what was right, and he did not mince words in saying it. One little anecdote will make this clear:

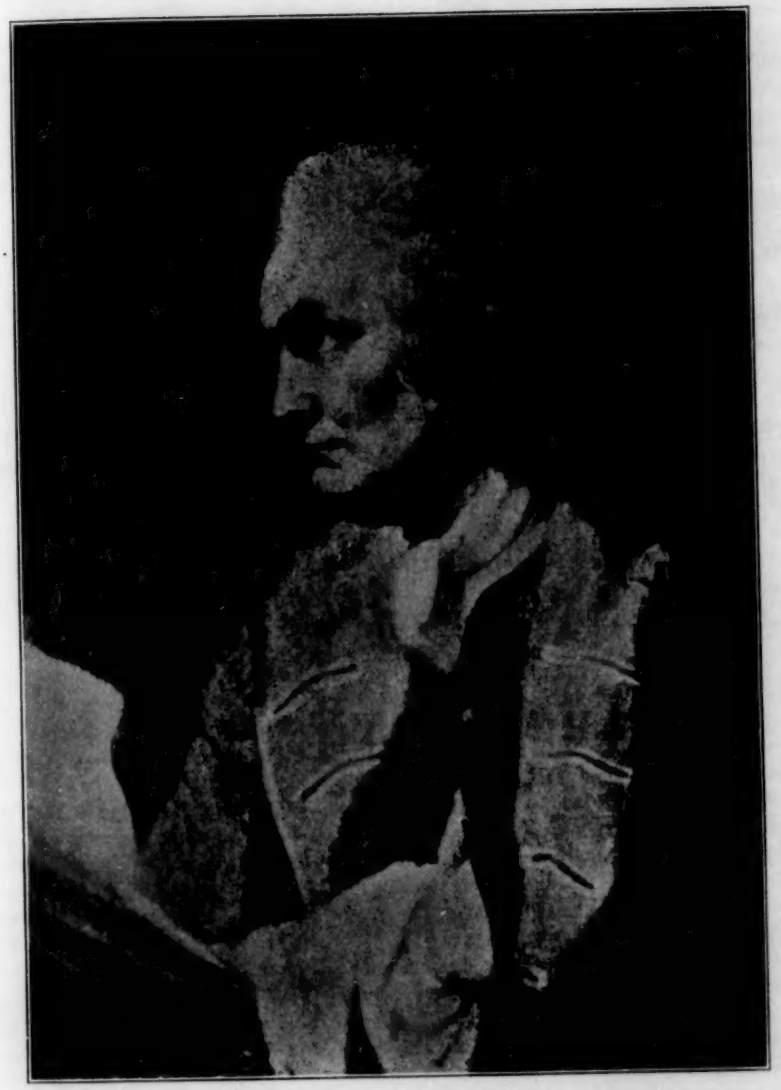
> A Lady of great sway among her acquaintance told me not long ago with an air of reproach, that she had nursed her Child according to my book, and it died. I asked, if she had suckled it herself? No. Had it sucked any other woman? It was dry-nursed. Then, Madam, you cannot impute your misfortune to my advice, for you have taken a method quite contrary to it, in the most capital point. Oh but, according to my direction, it had never worn stockings. Madam, Children may die, though they do or do not wear stockings.

The "Essay on Nursing" is a little pamphlet of forty-two octavo pages, published by the General Committee of the Foundling Hospital, and it was sold at sixpence a copy. It stars off in true Cadogan style:

> It is with great Pleasure I see at last the Preservation of Children become the Care of Men of Sense: It is certainly a matter which well deserves their Attention, and I doubt not, the Publick will soon find the good and great Effects of it.

The essay is well worth reading and should be reprinted somewhere for the benefit of the present generation, for it is like a fresh breeze. A few more extracts will give an idea of his point of view:

> In the lower class of Mankind, especially in the country, disease and mortality are not so frequent, either among adults or their children. Health and posterity are the portion of the poor, I mean the laborious. The want of superfluity confines them more within the limits of Nature: hence they enjoy blessings they feel not, and are ignorant of their cause. The mother who has only a few rags to cover her child loosely, and little more than her own breast to feed it, sees it healthy and strong, and very soon able to shift for itself; while the puny insect, the heir and hope of a rich family, lies languishing under a load of finery that overpowers his limbs, abhorring and rejecting the dainties he is crammed with, till he dies a victim to the mistaken care and tenderness of his fond Mother.
>
> When a man takes upon him to contradict received opinions and prejudices sanctified by time, it is expected he should bring valid proof of what he advances. The truth of what I say, that the treatment of Children in general is wrong,



WILLIAM CADOGAN

1711-1797

unreasonable, and unnatural, will in a great measure appear, if we but consider what a puny valetudinary race most of our people of condition are; chiefly owing to bad nursing, and bad habits contracted early. But let any one, who would be fully convinced of this matter, look over the BILLS OF MORTALITY; there he may observe, that almost half the number of those who fill up that black list, die under five years of age: so that half the people that come into the world, go out of it again before they become of the least use to it, or themselves. To me this seems to deserve serious consideration; and yet I cannot find, that any one man of sense and publick spirit has ever attended to it at all; notwithstanding the maxim in every one's mouth, that a multitude of inhabitants is the greatest strength and best support of a Commonwealth.

His credo is expressed in the following sentence:

You perceive, Sir, by the hints I have already dropped, what I am going to complain of is, that Children in general are over-cloathed and over-fed; and fed and cloathed improperly. To these causes I impute almost all their diseases.

He gave directions for clothing, for airing, for feeding and for the general management of infants and children, and it would take but little editing to make this essay fit for distribution at the present day. His diet was to add vegetables and fruit early. Note the four feedings a day in the extract below.

On the subject of allowing children to die from neglect as used to be, and alas still is the custom though notably lessened, he wrote as follows:

The Child, was it nursed this way, would be always quiet, in good humour, ever playing, laughing, or sleeping. In my opinion, a Man of sense cannot have a prettier rattle (for rattles he must have of one kind or other) than such a young Child. I am quite at a loss to account for the general practice of sending infants out of doors, to be suckled or dry-nursed by another Woman, who has not so much understanding, nor can have so much affection for it as the Parents; and how it comes to pass, that people of good sense and easy circumstances will not give themselves the pains to watch over the health and welfare of their Children; but are so careless as to give them up to the common methods, without considering how near it is to an equal chance, that they are destroyed by them. The ancient custom of exposing them to wild beasts, or drowning them, would certainly be a much quicker and more humane way of dispatching them. There are some, however, who wish to have Children, and to preserve them, but are mistaken in their cares about them. To such only would I address myself, and earnestly recommend it to every Father to have his Child nursed under his own eye, to make use of his own reason and sense in superintending and directing the management of it; nor suffer it to be made one of the mysteries of the Bona Dea, from which the Men are to be excluded. I would advise every Mother that can, for her own sake, as well as her Child's to suckle it. If she be a healthy Woman, it will confirm her health; if weakly, in most cases it will restore her. It need be no confinement to her, or abridgment of her time; four times in four and twenty hours will be often enough to give it suck; letting it have as much as it will take out of both breasts at each time. It may be fed and dressed by some handy reasonable servant, that will submit to be directed; whom likewise it

may sleep with. No other Woman's milk can be so good for her Child; and dry-nursing I look upon to be the most unnatural and dangerous method of all; and, according to my observation, not one in three survives it. To breed a Child in this artificial manner, requires more knowledge of Nature, and the animal economy, than the best Nurse was ever mistress of, as well as more care and attention than is generally bestowed on Children: the skill of a good Physician would be necessary to manage it rightly.

News and Comment

UNIVERSITY NEWS

Northwestern University Medical School plans to erect a children's hospital and clinic on McKinlock Campus, Chicago, as soon as funds are available. The estimated cost of the hospital will be about $1,500,000, with a capacity of 100 beds. The hospital will be under the jurisdiction of Dr. Isaac A. Abt, professor of pediatrics at Northwestern.

The Harvard University School of Medicine recently announced the appointment of Charles Fremont McKhann as assistant professor of pediatrics.

PERSONAL

The distinguished Brazilian professor of pediatrics, Olinto De Oliveria, has recently been made a member of the Society of Pediatrics of Paris.

GENERAL

Sixth Congress of French-Speaking Pediatricians.—The congress will be held at Strasbourg during the first six days of October, 1930, under the presidency of Dr. P. Rohmer. Problems for discussion at this conference will include: (1) the hilus of the lung in normal and diseased children; (2) fulminating and atypical disorders affecting the growth of infants; (3) alimentary fevers; (4) chronic types of nephritis in infancy; (5) diagnosis and treatment of cerebral tumors, and (6) treatment for the various forms of cerebrospinal meningitis.

Bequests and Donations.—The Roman Catholic Orphan Asylum and the New York Foundling Hospital receive $10,000 by the will of the late Samuel Stern, of New York.

The Children's Hospital, Cincinnati, is given $5,000 by the will of the late Judge Runyan, of Lebanon, Ohio.

White House Conference for Child Health and Protection.—The personnel of the committee on growth and development is as follows:

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Abstracts from Current Literature

Pathology, Bacteriology and Biochemistry

BRUCELLA ABORTUS IN MILK SUPPLY AS A SOURCE OF AGGLUTININS IN HUMAN SERA. MERRILL J. KING and DOROTHY W. CALDWELL, Am. J. M. Sc. **178**:115, 1929.

Persons with lowered resistance who drink raw milk infected with *Brucella abortus* may develop agglutinins in their blood serum with or without the manifestation of appreciable clinical symptoms of undulant fever. Of 851 patients and 156 staff members in a sanatorium in which raw milk was used, 91, or 9 per cent, showed abortus agglutinins when their serums were diluted 1:15 or higher. Twenty-four of the ninety-one had agglutinin titers varying from 1 to 45 to 1 to 3,200. The presence of abortus agglutinins in human serum is evidence of infection with *Brucella abortus*. The agglutinins may persist in the serum for months or years after the recovery of the patient. There is complete lack of evidence of porcine infection in the herd studied. The correlation between the periods of *Brucella abortus* infection of the milk supply and the occurrence of agglutinins in the serums of the patients indicates a bovine origin of the cases of undulant fever reported in this study. *Brucella abortus* may be present in small numbers in the milk of infected cows, but the organisms may be eliminated for several years. The presence of *Brucella abortus* agglutinins in the blood serums of cows does not determine whether they are discharging *Brucella abortus* in their milk. In a herd of 151 animals, no evidence was obtained of the infection of the milk of cows whose serums agglutinated at 1 to 60, while cultures of *Brucella abortus* were obtained from the milk of only 23 of 56 cows with titers of 1 to 120 or higher. *Brucella abortus* was not isolated from the blood or from the urine of the infected cows.

AUTHORS' SUMMARY [ARCH. PATH.].

AN UNUSUAL TYPE OF TRIATRIAL HEART. B. M. PATTEN and W. B. TAGGART, Arch. Path. **8**:894 (Dec.) 1929.

A white female infant who had shown rapid respiration and cyanosis since birth died at the age of 3 months. Autopsy showed that nine tenths of the blood returning from the lungs entered an accessory median atrium which was virtually cut off from the left side of the heart. From this median chamber the blood passed through a large septal defect into the right atrium. Thence it was distributed between the enlarged right ventricle, which must have received the major share, and the left atrium, which could receive mixed blood through an open foramen ovale. The mixed blood entering the left atrium was enriched by a small quantity of fully oxygenated blood from the right superior pulmonary vein. There was also a patent ductus arteriosus which may have been of slight help in relieving the oxygen deficiency of the systemic circulation. The authors give an embryologic interpretation and discuss related cases. The article is well illustrated.

LESLIE, Evanston, Ill.

CREATINE-CREATININE EXCRETION AND SERUM CALCIUM IN NORMAL MALE CHILDREN. M. KARSHAN, R. G. FREEMAN, JR., and E. G. MILLER, JR., Arch. Pediat. **46**:323 (May) 1929.

Data were obtained in a series of normal boys, from 5 to 9 years of age. The creatine of the urine was determined by the Benedict method, and the creatinine by the method of Folin. The children were "clinically normal." The serum calcium was determined by the Tisdall modification of the Kramer-Tisdall method.

The authors state that there is no evidence of any significant difference in any of the series as regards the age of the subjects from 5 to 9 years. The magnitude of the deviations in the normal, especially in the case of urinary creatine and creatinine, suggests that diagnosis of abnormality of metabolism by these analyses should be undertaken with considerable caution. The day to day variation of the creatine-creatinine metabolism in a given child was as great as the variation between different children.

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FURTHER NOTES ON THE FILTRATION OF THE VIRUS OF VACCINIA. HUGH K. WARD, J. Exper. Med. **50**:31, 1929.

An active filtrate can be obtained from vaccinia lesions by grinding up the fresh tissue with glass fragments, emulsifying in hormone broth, centrifuging the emulsion and filtering the supernatant fluid through a Berkefeld V filter. The sterile filtrate so obtained has been shown by comparative titration on rabbits to have about one sixteenth of the activity of the nonsterile emulsion used in vaccination of human beings. Centrifugation of such a filtrate shows a partial concentration of the virus in the lowermost layer. The virus survives for a long time, if the filtrate is kept near the freezing point, and probably will survive indefinitely if kept frozen. The addition of glycerin is not necessary.

AUTHOR'S SUMMARY [ARCH. PATH.].

RELATION OF VACCINAL IMMUNITY TO THE PERSISTENCE OF THE VIRUS IN RABBITS. PETER K. OLITSKY and PERRIN H. LONG, J. Exper. Med. **50**:263, 1929.

By means of cataphoresis, vaccine virus can be obtained from suspensions of tissue which are inactive by the usual tests of animal inoculation. Active virus can be obtained by cataphoresis from the tissues of rabbits long recovered from the effects of cutaneous vaccination. Evidence is brought forward which suggests that immunity in a virus disease, such as vaccinia, may be linked with the persistence of living virus in the body.

AUTHORS' SUMMARY [ARCH. PATH.].

THE EFFECT OF CATAPHORESIS ON POLIOMYELITIS VIRUS. P. K. OLITSKY, C. P. RHOADS and P. H. LONG, J. Exper. Med. **50**:273, 1929.

Under ordinary conditions of hydrogen ion concentration the virus of poliomyelitis, as such, or associated with particles in fine suspensions, migrates in an electrical field to the anode. It follows that the virus bears an electronegative charge. By means of cataphoresis, the virus can be recovered from a noninfective mixture of virus and specific immune serum. By the same means it is possible to reveal the presence of virus in the central nervous system of a monkey which has recovered from the active stage of experimental poliomyelitis.

AUTHORS' SUMMARY [ARCH. PATH.].

ADAPTATION OF MASTITIS STREPTOCOCCI TO MILK. F. S. JONES and H. S. SIMMS, J. Exper. Med. **50**:279, 1929.

The data here presented indicate that the inhibitory principle affecting the growth of streptococci in milk is not greatly utilized during the lag phase, and that the abrupt termination of lag is not due to the utilization of the principle. They further indicate that the sudden beginning of growth cannot be ascribed to a resistant type of streptococcus present in the culture from the first, but to an adaptation occurring during the lag phase. The addition of large numbers of dead or living streptococci to milk, for limited periods, fails to diminish appreciably in the inhibitory principle.

AUTHORS' SUMMARY [ARCH. PATH.].

THE REACTION OF VARIOLA VACCINE-VIRUS IN TISSUE CULTURE. E. HAAGEN, Centralbl. f. Bakteriol. **109**:31, 1928.

Vaccine virus in rabbit-testis tissue culture developed well provided it was freshly explanted every four or five days. The virulence of the virus cultivated in this manner for eight months through thirty-seven passages remained for the most part unchanged, and in a few cases, even increased.

PAUL R. CANNON [ARCH. PATH.].

THE COLLOIDAL OSMOTIC PRESSURE OF THE BLOOD IN INFANCY. A. MAZZEO, Monatschr. f. Kinderh. **45**:318 (Nov.) 1929.

For determining the colloidal osmotic pressure of blood serum, the writer used the apparatus of Krogh and Nakazawa which has the advantage of being inexpensive and needing only small amounts of blood. He then made determinations in normal children and children suffering from febrile infections, anemias, pneumonia and empyema, and syphilis. He was not able to draw any clinical conclusions concerning the relations of the colloidal osmotic pressure to these diseases.

GERSTLEY, Chicago.

ANAPHYLAXIS AND ACID-BASE BALANCE. STEPHAN VON PELLÁTHY and EDMUND HAINTZ, Wien. klin. Wchnschr. **42**:1408 (Oct. 31) 1929.

Anaphylactic shock is increased by any condition which increases vagal tone, such as acidosis, and is decreased by any condition which increases sympathetic tone, such as alkalosis. The authors' experiments with animals sensitized with horse-serum show that previous administration of 6 cc. of 8 per cent sodium bicarbonate by mouth for three days lessened the mortality from anaphylactic shock (a mortality of 28.5 per cent; in controls, 50 per cent). The administration of 6 cc. of fifth-normal hydrochloric acid for three days increased the mortality to 62.5 per cent. If, however, a single dose of alkali is given simultaneously with the shocking dose of protein, no benefit is apparent.

CLAUSEN, Rochester, N. Y.

THE PHYSIOLOGY OF DIGESTION IN THE INFANT. O. BUDDE, Ztschr. f. Kinderh. **47**:486 (May) 1929.

In the stomach of the infant after feeding with milk there is a splitting of protein; this is less in human than in cow's milk. The curves of p_H activation in relation to the influence of pepsin on skimmed milk expressed in units of time for a conversion of the same degree resemble extensively the curves obtained by Sörensen with acid albumin. Complete absence of the splitting of protein is not to be found even between p_H 5 and 6. For the same unit of time the conversion of skimmed milk or of casein solution brought about by pepsin is distinctly higher than a solution of egg or of cow's milk albumin of like concentration. As opposed to solutions of albumin, the optimum activity seems in cow's milk and solutions of casein to be pushed toward the alkaline side. In human milk less nitrogen is split off by solutions of pepsin and by the gastric juice of infants at the same p_H than in one-third cow's milk. This observation is explained by the greater content in human milk of albumin which is attacked with difficulty. This also explains the fact that human milk is less well split in the stomach of the infant.

EMERSON, Boston.

PRECIPITATION OF BLOOD PROTEIN FRACTIONS. K. ŠVEHLA and Č. SAITZ, Časop. lék. česk. **66**:216 (Feb. 4) 1927; 264 (Feb. 11) 1927.

The authors discuss the attempt to establish a norm for the various protein fractions in various conditions, such as nephritis, icterus, diabetes, tuberculosis,

syphilis, anemia, leukemia, intoxication, decomposition, etc. Absolute alcohol, magnesium sulphate and calcium chloride are used according to the methods of Ruzicka and Matefy. Increased flocculability of serum is frequently found in syphilis, tuberculosis and progressive atrophy. An indefinite increase is found in prematurity, icterus neonatorum, severe respiratory infections and ante mortem. Increased precipitation is found as one of the symptoms of exudative diathesis, possibly on a constitutional basis. Increased precipitation may be related in some way to the associated eosinophilia, as in exudative diathesis, and may have some bearing on the outcome of exudative diathesis associated with syphilis or tuberculosis.

STULIK, Chicago.

BLOOD VOLUME IN HEALTHY CHILDREN. P. KISS and Z. TEVELI, Orvosi hetil. **73**:816 (Aug. 17) 1929.

Blood determinations were made on healthy children of ages between 2 and 10 years. The method used was the injection of brilliant vital red. The absolute blood volume per kilogram of body weight shows from 43.3 to 66.3 cc. of plasma, and from 80 to 1,225 cc. of blood. The total amount of blood ranges between 1,130 and 3,417 cc. The absolute amount of plasma ranges from 690.5 to 2,083.0 cc.

ZENTAY, St. Louis.

CONNECTION BETWEEN ANAPHYLAXIS AND ACID BASE EQUILIBRIUM. I. PELLATHY and O. HAINTZ, Orvosi hetil. **73**:1076 (Oct. 26) 1929.

The authors studied the connection between anaphylactic shock and acid base equilibrium in a series of experiments on guinea-pigs. The animals were divided into three groups. The first group was that of the control animals. The second group received through a stomach tube a 6 per cent solution of sodium bicarbonate three days before sensitization and also every day until the reinjection. The third group received 6 cc. of fifth-normal hydrochloric acid solution in the same manner. The diets of the animals were the same in all three groups. Sensitization was produced with 0.01 cc. of normal horse serum. After three weeks, 0.02 cc. of serum per hundred grams of body weight was injected into the jugular vein. This amount is about the minimum lethal dose. Results of the experiments in this series are: Fifty per cent of the untreated animals died under the shock. The alkalinized animals showed a mortality of 28.5 per cent, while the acidotic animals showed a loss of 62.5 per cent. In another series, the animals did not receive any acid or alkali before the experiment or during sensitization. At the time of reinjection the first group received 1 cc. of physiologic saline solution. The second group received 1 cc. of a 5 per cent solution of sodium bicarbonate. The third group received 10 cc. of an acid phosphate solution containing also 1 per cent tenth-normal hydrochloric acid. The results of this series are: Of the normal animals 22.2 per cent died, as did 22.5 per cent of the alkalotic and 66.6 per cent of the acidotic animals. It seems that the change of the p_H in the acidotic direction increases the anaphylactic shock, while a change in the alkalotic direction decreases the intensity of the shock.

ZENTAY, St. Louis.

Nutrition

CLINICAL SIGNIFICANCE OF THE WATER OF METABOLISM IN CHILDREN. HYMAN GOLDSTEIN, Arch. Pediat. **46**:371 (June) 1929.

The human body is compared to a huge chemical laboratory with an important irrigation system. The importance of water in its physiologic contribution to the human machine is discussed at length. A proper water balance is essential to health. Dehydration results from a marked negative balance. If the water balance is strongly to the positive side, edema results.

The water metabolism is an important factor and is of great clinical significance in children in health and in diseased conditions. Diet modifies the water balance a great deal. Carbohydrates and protein favor water retention; but in excess, especially if protein is over 20 per cent of the total calories, metabolism and water loss is stimulated and hence there is weight loss. If carbohydrate is less than 3.5 per cent of the diet, acidosis may result. A high fat diet encourages water loss and indirectly weight loss. Alkalosis may result from severe vomiting with loss of water and hydrochloric acid.

WILSON, Norfolk, Va.

HYPEREMESIS HIEMIS OR THE WINTER VOMITING DISEASE. JOHN ZAHORSKY, Arch. Pediat. **46**:391 (June) 1929.

The disease occurs in widespread epidemics in the fall and winter months. It does not occur in summer. One of the first epidemics seen was in 1904 when the author treated thirty patients. It occurred in October and November and was not associated, except in a few instances, with an acute respiratory infection. The epidemics last from two to four weeks, rarely longer; the last one occurred in 1929. The disease occurs mostly in infants and very young children. Occasionally it is seen in older children and in adults. Breast-fed infants are generally immune.

The symptoms often begin suddenly, but frequently the older child complains of abdominal pains for one or two days preceding an acute attack. Many infants are peevish and restless during the night before the vomiting, which is violent and forcible and often accompanied by symptoms of collapse. This continues for one or more days; everything swallowed is vomited. Within a few hours the bowels become loose and continue loose for a period of from a few hours to three days. Diarrhea is not always present. The stools are thin and very light in color, and have an offensive odor. Excoriation of the skin about the anus is often found. Prostration is the rule. The child is listless for from one to three days, and is sometimes in a semicomatose or stuporous state.

The temperature is not high (from 99 to 101 F.). The severe cases show acidosis after a time. A few cases give signs of dehydration, but typical gastroenteric intoxication is rare. Recovery invariably occurs after a few days. The author has not seen a single death, but he mentions several cases in which the symptoms were so severe as to cause the greatest anxiety. Physical examination shows nothing of importance. Pallor and prostration are obvious. There is no abdominal tenderness and only exceptionally is distention present. The pulse may be feeble; hyperpnea or acidosis may be present. The skin retains its normal elasticity. The urine is scanty and may contain a trace of albumin. The stools show no pus cells. The leukocyte count varies from 7,000 to 13,000.

The diagnosis is based on: (1) the presence of an epidemic, (2) the afebrile course, (3) the persistent vomiting and the peculiar offensive light-colored stools not due to an inflammatory condition of the bowels and (4) the absence of an acute respiratory infection (the fourth condition is difficult to fulfil in many cases in the winter). For purposes of comparison, a case of allergic enteritis, one of grippal enteritis, one of influenza gastritis and one of vomiting disease are described in detail.

Nothing positive is known as to etiology. The following theories have been considered: 1. The disease is caused by ingestion of milk that has been frozen. 2. The disease is a peculiar manifestation of influenza. 3. A respiratory infection, commonly called grip, is the source. 4. The disease is caused by some toxic substance in milk, cream or butter. While the author favors the last-named theory, it does not account for all the facts. The name proposed is "winter vomiting disease."

WILSON, Norfolk, Va.

GROWTH OF PRIVATE DAY SCHOOL BOYS. R. G. FREEMAN, JR., and RAYMOND SEARFOSS, Arch. Pediat. **46**:450 (July) 1929.

Tables are published showing the difference in growth of about 200 scholars from favored environments in a private day school in New York, as compared with Gray's series of boarding school boys, Baldwin's gifted children and Bow-

ditch's Boston series. The tables show that the private school boys are taller than the public school boys, and that the private day scholars are taller than those in Gray's series. From the tables it is seen that there are two major phases of accelerated growth; one from 11 to 12 years and one from 14 to 14½ years, with a minor phase at 7 years.

WILSON, Norfolk, Va.

RICKETS. C. E. MACKE and W. J. TODD, Arch Pediat. **44**:465 (July) 1929.

A small percentage of cases shows no roentgen or other evidence of bony changes. The patient exhibits other evidences of rickets. In the presence of syphilitic activity there is marked lowering of the blood calcium and phosphorus; the percentage is much lower than when rickets is present alone. Such children often show no evidence of syphilis. It is much more difficult to secure improvement in the rachitic process of such children than in those whose condition is not complicated by syphilis.

WILSON, Norfolk, Va.

BERIBERI IN LATE INFANCY: THE RESULT OF CELIAC DISEASE. SIDNEY V. HAAS, Arch. Pediat. **46**:467 (Aug.) 1929.

Beriberi is produced by a deficiency of vitamin B, but may be due to an inability of the organism to abstract this substance from the ingested food. Beriberi is commonly seen in the orient but it may occur anywhere.

The great similarity of celiac disease to beriberi suggests that they may be one and the same disease; for the following reasons the author concludes that they are not: 1. Many cases of celiac disease presented none of the symptoms of beriberi. 2. Beriberi was supposed not to occur in this country except in rare instances. 3. Feeding a diet adequate in vitamin B did not relieve the symptoms. 4. Even a symptomatic cure with a diet of bananas did not at once overcome the intolerance for other forms of carbohydrate and some of the fats, the characteristic fault in celiac disease.

Beriberi results from faulty metabolism directly caused by a deficiency of vitamin B. Celiac disease is characterized by inability to utilize carbohydrates and fats, the chief difficulty being with carbohydrates, and hence the person is unable to secure sufficient vitamin B even though the diet is particularly rich in this substance.

The pathology and symptoms of the two diseases are compared and cases illustrating their striking likeness are reported. The difference lies in the degeneration of the nervous system in beriberi. The author is not aware of any postmortem reports on the nervous system in celiac disease, but concludes that the paralyses in celiac disease and the response to a food rich in vitamin B seem to show definitely that celiac disease may terminate in beriberi.

WILSON, Norfolk, Va.

A STUDY OF FOOD CONSUMPTION OF CONVALESCING CHILDREN. A LEVINSON, K. MITCHELL THOMA and NELL TAYLOR, Arch. Pediat. **46**:487 (Aug.) 1929.

Three groups of children were studied. The first group comprised 3 boys observed for a period of one month. The second group was composed of 23 boys and 10 girls, observed for four months, and the third group of 87 boys and girls observed over a period of six months, a total of 123 convalescing children. They did not eat everything served. The consumption of milk averaged 750 Gm. daily per patient, supplying sufficient calcium. The proteins eaten were sufficient to supply from 1.5 to 2 Gm. per kilogram of body weight. All vitamins were supplied. The amount of iron was small and may have been insufficient. The total caloric intake was apparently adequate. A large percentage of the children had to be taught to eat vegetables. More of them liked lettuce than liked the cooked vegetables. The children ate better when under supervision.

WILSON, Norfolk, Va.

REGULATED AND UNREGULATED OVERFEEDING IN THE GENESIS OF RICKETS AND SCORBUTUS. MOURIQUAND, Arch. de méd. d. enf. **31**:395, 1929.

The writer has previously shown the ill effects of regulated and unregulated underfeeding and has demonstrated that underfeeding of bottle-fed infants commonly provokes major dystrophies, the gravest expression of which is represented by the athrepsia of Parrot which may result fatally in patients under the age of 5 months. Beyond this period, underfeeding is apt to be more commonly exemplified in hypotrophic disturbances. Mouriquand concluded from this study that hypo-alimentation, with dystrophy, was only rarely accompanied by the classic features of rickets, and that, similarly, even those infants suffering from underfeeding and from a deficiency of vitamin C rarely developed scurvy.

It is true that in many cases of hypothrepsy and hypotrophy, as reported by Marfan and Variot, in which young infants are developing slowly or not at all, some of the classic signs of rickets may appear, such as deformities of the cranial bones and malformation of the chest. These are found especially in patients suffering from digestive disturbances and hereditary syphilis, and from lack of sunlight. The more florid manifestations of rickets, however, such as intumescence of the extremities, involvement of the epiphyses, rachitic rosary, late closure of the fontanel and tardy locomotion, are usually observed in infants beyond the age of 6 months who are well nourished or actually overnourished, particularly when indigestion and lack of sunlight are accompanying factors. In rats, experimental studies have proved that rickets does not develop except during the stage of growth. Mouriquand previously reported his opinion that generalized rickets is found only during those periods of most rapid growth, late infancy and puberty, and that it tends to recede during the more quiescent periods of development. If the normal gain can be reduced by lower caloric intake or by the use of thyroid (Mouriquand, Leulier, Schoen), the progress of rickets in experimental cases can be arrested. It is recognized that, so far as etiology is concerned, experimental rickets does not approach in all respects the human type, but it is maintained that in both cases accelerated growth of the organism is a factor in the development of the syndrome termed rachitis. It becomes necessary, then, to distinguish the effect of regulated superalimentation from that of promiscuous overfeeding. The best illustration of the former is the overfed baby at the breast, in whom the excessive intake, more than counterbalanced by the normal ingredients of human milk — albumins, salts, sugars, fats, vitamins and enzymes — is rarely followed by grave digestive disturbances or serious dystrophies. In spite of the overload the digestive tube does not commonly rebel, and the infant simply surpasses the physiologic requirements as to weight, strength and energy. Free from infections, these infants practically never exhibit any of the major signs of rickets; alimentary equilibrium is maintained under the "specificity" of mother's milk.

This does not obtain in artificial feeding unless carefully supervised; the rôle of superalimentation, with an ill balanced and unregulated ration, in bottle-fed infants has no less important a bearing on the origin of grave dystrophies than has underfeeding. Digestion is obtunded, important secretions are diminished and toxic products are produced and absorbed; a terrain is found that is suitable for the development of rickets. If abstention from sunlight is added to these factors, the onset of rickets is hastened, provided the infant is increasing in weight.

Scorbutus is usually considered as coexistent with inanition, appearing as it does among besieged populations, in armies on retreat, etc. However this may be among adults, Comby and others have made it clear that in infants served with a ration low in vitamin C for long periods, the alimentary origin of scurvy is evident. In certain cases it would appear that intercurrent infections, such as grip (Hess), hastened the onset. Mouriquand's observations lead him to believe that the great majority of patients with scorbutus are found not among the undernourished infants but in those who are overfed and gaining normally in weight. Infants over 6 months of age on a restricted ration with only a minimum allowance

of vitamin C rarely develop signs of scurvy. Guinea-pigs fed ad libitum on barley and hay will develop scorbutus about the twelfth day, the lesions rapidly accentuating themselves for the next two or three weeks, when death ensues. It is notable that in spite of the osteomuscular manifestations and hemorrhages in the joints the animal continues to gain in weight, its appetite undisturbed, up to about the third week. If, however, one half or one third of the ration is allowed, the animals cease to gain in weight but do not develop scurvy, as shown at autopsy. These experiments were carried on during the winter. Similar investigations made during the summer occasionally were followed by the appearance of scurvy in a starved animal; this was interpreted as evidence in favor of numbering heat among the etiologic factors.

AMESSE, Denver.

MEAT IN THE INFANT DIET. A. B. MARFAN, Nourrisson **17**:149 (May) 1929.

Marfan disapproves of meat or meat extractives in the diet of healthy infants under the age of 1 year. After this age, meat is permitted in the form of soups or panade. As soon as the infant is able to masticate, he allows meat that is ground or cut into small pieces.

The indications for giving meat in any form to sick infants under 1 year of age are few. In convalescence from enterocolitis, in celiac disease, in tuberculosis and in nutritional anemias, the addition of meat that is raw or cooked is often helpful. The advisability of giving meat to young infants who have an idiosyncrasy to milk and to those suffering from infantile eczema is also discussed.

McCLELLAND, Cleveland.

PANCREATIC FERMENTS IN DIGESTIVE DISTURBANCES. S. STROE, Nourrisson **17**:193 (July) 1929.

The author describes his method of obtaining and evaluating the potency of the external secretion of the pancreas in infants, and the alterations encountered in the common digestive disturbances.

In the severe diarrheas one finds a marked lowering of all the ferments, amylase and trypsin being reduced 50 per cent and lipase 70 per cent. Cases of hypothrepsia and athrepsia show smaller reductions from normal figures. In the less severe diarrheas, in which the patients have four or five stools per day, without fever and without constitutional reactions, the values depart only slightly from the normal, the most marked lowering affecting lipase only. The information gained in this way can be utilized in prescribing diets appropriate to the impaired digestive functions.

McCLELLAND, Cleveland.

IRRADIATED ERGOSTEROL IN THE TREATMENT FOR RICKETS AND TETANY. A. B. MARFAN and ELISABETH DOLLFUS-ODIER, Nourrisson **17**:205 (July) 1929.

Reference is made to the researches of A. Hess, Steenbock and Black, Rosenheim and Windaus. In their own cases the authors found that irradiated ergosterol produced a rapid calcifying of the diseased bones and a prompt disappearance of signs of tetany. The figures for the blood calcium and cholesterol were quickly raised to the normal. Much less striking effects were obtained on the other manifestations, such as muscular hypotonia, lymphoid hyperplasia and anemia. The latter seem to respond better to the direct ultraviolet therapy. The ease of administration and convenience commend the use of irradiated ergosterol, however.

McCLELLAND, Cleveland.

MODE OF ACTION OF IRRADIATED ERGOSTEROL IN RICKETS. A. B. MARFAN, Nourrisson **17**:213 (July) 1929.

The fact that ultraviolet rays and irradiated ergosterol affect favorably the bone lesions in rickets does not convince Marfan that rickets is due merely to

a lack of these factors. He believes that the genesis of the disease is more profound. In his opinion, chronic infections and intoxications assume the major rôles. Congential syphilis, tuberculosis (especially scrofula), prolonged bronchopneumonia, pyodermia and occult diphtheritic foci are the infections which he believes should be incriminated. The chronic intoxications he blames on defective alimentation and chronic digestive disturbances. These infections and intoxications are able to prevent activation of the ergosterol present in the skin and organs of infants so diseased, resulting in the train of symptoms recognized as rickets. He looks on the bone lesions and deformities as merely minor manifestations of a generalized malady. Attention is called to the fact that rickets is rare in Greenland in spite of the prolonged absence of the sun during the winter months. This rarity of rickets is credited to the fact that breast feeding there is the common practice and that the nursing mothers there consume large quantities of cod liver oil or seal oil, both of which are rich in irradiated ergosterol which, in turn, is eliminated through the mammary glands. Furthermore, syphilis is practically nonexistent in Greenland.

McClelland, Cleveland.

The Quantity of Iron in Breast Milk. Dorlencourt and Calugareanu-Nandris, Nourrisson **17**:227 (July) 1929.

The average amount of iron per liter of breast milk was found to be 3 mg., the extremes being 1.4 and 7.07 mg. Considerable variation was found in samples taken at different times of the day; likewise in samples from the right and left breasts, respectively. There was no consistency in these observations, so it was not possible to make any deductions. The age of the mother seemed to bear some relation to the iron content. Young mothers, aged 18 or 19, averaged about 2 mg. per liter. The amount increased with age to a maximum of 4.01 mg. at 27, and then decreased again to 2.51 during the fourth decade. The duration of lactation did not appear to bear any direct relation to the iron content of the milk secreted. The comparison of observations on primiparas and multiparas varied little (2.35 and 2.77 mg.). Finally experiments were made to determine the effect on the iron content of breast milk by giving soluble iron salts by mouth to the mother. Definite increases were obtained. The authors concluded that the mothers of anemic infants should receive iron therapy.

McClelland, Cleveland.

Hypo-Alimentation. Jean Lacroix, Thèse de Lyon, 1929.

The author discussed 186 cases of hypo-alimentation which have come under his observation. Infants receiving breast milk, though markedly underfed, have a much better chance of rehabilitation than those receiving a poorly balanced diet with insufficient protein. Frequently the latter group has been fed largely on vegetable soups and cereals. Contrary to general opinion, diarrhea and vomiting are frequent symptoms. The younger the infant, the more severe is the condition which may be brought about, showing that the rapidly growing organism is particularly susceptible to injury (atrophy and decomposition). In these cases a well balanced food is essential; breast milk should be supplied whenever possible.

Leslie, Evanston, Ill.

Mammary Gland and Amino-Acid. P. Bosio, Riv. di clin. pediat. **27**:515 (May 30) 1929.

The author found that the percentage of amino-acid in blood drawn from the mammary vein of the lactating sheep was, on an average, 23 per cent less than that in blood from the tibial vein or the jugular vein, and 33 per cent less than that in blood from the femoral artery. The lactating mammary gland thus retains more amino-acid than the other body tissues, as would be expected since milk protein is synthesized there.

Higgins, Bostor.

THE EUTROPHIC ACTION OF IRRADIATED FOODS. L. SPOLVERINI, Riv. di clin. pediat. **27**:803 (Nov.) 1929.

The author favors the use of irradiated milk throughout the first two years of life in preference to viosterol. He feels that it is better if only some of the food, and not all of it, is irradiated.

HIGGINS, Boston.

EFFECT OF ERGOSTEROL ON THE AORTA OF RABBITS. R. MANCKE, Arch. f. exper. Path. u. Pharmakol. **141**:228, 1929.

On the addition of irradiated ergosterol to the food of rabbits, marked calcification of the aorta with aneurysmal dilatation resulted regularly.

[ARCH. PATH.]

IRRADIATED FOODS AND ERGOSTEROL IN THE TREATMENT FOR RICKETS AT THE BASEL CHILDREN'S CLINIC. E. WIELAND, Deutsche med. Wchnschr. **55**:1296 (Aug. 2) 1929.

After verifying the results reported by American investigators with the use of irradiated substances in the treatment for rickets, an irradiated milk was prepared at the author's clinic in Basel, in 1925, and was used for feeding rachitic infants. A satisfactory dried milk was available and this was activated by quartz light. Irradiated fresh milk was also used but found to be rather cumbersome in its preparation. Proprietary irradiated ergosterol preparations, in both oil and tablet form, were used in infants in which whole milk was contraindicated. It is notable that in these preparations no vitamin D strength is given, as is required for the American products.

After discussing the possibility of vitamin D poisoning, the author draws his conclusions from his experience with these preparations: (1) Irradiated ergosterol should never be given in any condition except rickets and its associated diseases, tetany, late rickets and osteomalacia. (2) The dose should never exceed from 2 to 4 mg. of activated ergosterol, from 80 to 85 Gm. activated dry milk, or 0.5 liter activated fresh milk. (3) The treatment should not be continued for more than from eight to ten weeks in uncomplicated cases of rickets.

SCHERER, Duluth, Minn.

USE OF OLIVE OIL IN FEEDING OF INFANTS AND CHILDREN. A. REUSS, Med. Klin. **25**:1733 (Nov. 8) 1929.

There is a pronounced prejudice against the use of vegetable oils as food in Central Europe, but in spite of this fact, pure olive oil seems to be a satisfactory food and can be used without any difficulty in infant and child feeding. The caloric value of olive oil is high. It is practically identical with 100 per cent fat content. It is easily assimilated by infants, as is shown in the experiments of Schick and his co-workers. Chemically it is closely related to body fats, and this accounts for the easy assimilation. The author gives a number of recipes in which olive oil can be used as the prinicpal ingredient. He also claims that children took the different dishes prepared with olive oil without any difficulty.

ZENTAY, St. Louis.

THE ULTRAVIOLET RAY TREATMENT FOR RICKETS. S. ROSENBAUM, Monatschr. f. Kinderh. **45**:242 (Nov.) 1929.

The paper is rather a review of the literature than an original contribution. The wave lengths that are most effective against rickets are those near 3,020 angstrom units. Waves under 2,900 units which do not exist in the solar spectrum may be more potent than the most effective sun rays. The good effect of treatment is independent of erythema or of pigmentation; but of course a lamp which is not strong enough to produce a pigmentation would probably be ineffective against rickets.

The effect of treatment can be estimated scientifically by observation of the osseous system, for instance, for craniotabes and the size of the fontanel; by roentgenograms, particularly of the wrist, and by estimations of blood phosphorus.

The writer then discusses the efficiency of various types of lamps used in Germany. He finds the carbon arc satisfactory. There is as yet no simple means of estimating the efficiency of a lamp.

Treatment should be given from two to three times a week for from one to three months. The combination of ultraviolet irradiation with cod liver oil and viosterol has no advantage.

Little injury can ever come from the routine treatment. Occasionally bronchitis has been reported.

GERSTLEY, Chicago.

PERIODIC VOMITING AND KETOSIS IN CHILDREN. L. SALOMONSEN, Acta paediat., suppl. 1, **9**:1 (Dec. 10) 1929.

Since recent theories concerning the mechanism of attacks of periodic vomiting have postulated an abnormal intermediary metabolism, chiefly of fat and carbohydrate, and since certain investigators, especially in America, have reported evidence of this abnormality not only during attacks but also in the free intervals, the author undertook comprehensive experiments on metabolism to throw more light on these questions. It is agreed that in an attack the carbohydrate reserves are rapidly exhausted; and from the behavior of the blood sugar curve during a short starvation period, the development of ketosis on ketogenic diets, and the fact that attacks are reported as having been precipitated by altering the fat and carbohydrate content of the diet, several investigators have claimed that even between attacks these patients show a tendency to burn carbohydrate more rapidly than normal, so that they quickly become susceptible to a ketogenic acidosis.

Fifteen normal children were starved, and their blood sugar, acetone, fat and urinary output of ketone bodies and nitrogen were observed. The blood sugar fell sharply in the first ten hours, then more slowly, reaching a minimum point from about thirty-six to forty hours after the last food intake. The blood acetone curve rose sharply at first, reaching a maximum on the second or third day of fast. The blood fat rose very slightly in the first two days. Short granular casts appeared in the urine in one or two days in most cases, and many patients showed moderately severe vomiting. The author compares these results with those reported by Ross and Josephs in patients subject to cyclic vomiting; on this basis he interprets the latter as within normal limits. (Unfortunately, the age of the author's patients, between 8 months and 4 years, largely vitiates such a comparison.)

The response of twelve normal children to a sudden transition from a normal to a ketogenic diet was then compared to that of six children subject to periodic vomiting. In the normal group there was a wide variation of response: The sturdy, placid ones were scarcely disturbed, while the more delicate, mercurial ones were thrown into a condition which in all respects except the violence and frequency of vomiting entirely resembled an attack of true periodic vomiting. The patients subject to periodic vomiting fared no worse than the most susceptible controls, though being all of the thin, nervous type they all showed considerable disturbance. In no case, however, was a true attack of typical cyclic vomiting precipitated.

A similar comparison was made between patients subject to cyclic vomiting and normal controls in respect to their response to the addition of large amounts of fat to a normal diet, and their response to the production of fever (by vaccination or by injections of a sulphur preparation). Here again, no differences could be detected between the behavior of the normal children who reacted more severely and those in the group with cyclic vomiting.

The return of blood sugar to normal and the disappearance of ketosis both in prolonged starvation and in the recovery from attacks of cyclic vomiting have previously been adduced as evidence of the formation of carbohydrate from fat. The evidence that such a transformation does not occur in adults does not neces-

sarily apply in the case of children. To the normal children previously studied, large increments of fat were given, sometimes in addition to a ketogenic diet, and at other times as the only food. The response of the blood sugar curve and the urinary excretion of ketone bodies convince the author that under these conditions sugar is formed from fat. On the other hand, the behavior of patients subject to cyclic vomiting but studied in the interval between attacks does not differ from the normal.

The author concludes that periodic vomiting does not represent a true metabolic abnormality. He places responsibility rather on factors of constitution and temperament. The precipitating causes of attacks are likewise incompletely understood. One must admit the possibility that purely nervous factors are sometimes determinant.

A valuable bibliography is appended.

McINTOSH, Baltimore.

STORING OF IRRADIATED ERGOSTEROL IN THE BODY. G. PETENYI, Orvosi hetil. **73**: 871 (Aug. 31) 1929.

Storing of vitamins in the form of a reserve in the body seems to be a general phenomenon for all the different kinds of vitamins. On the basis of his clinical experiments the author thinks that the antirachitic vitamin D can also be stored in the body. He gave a large dose of irradiated ergosterol to children with severe rickets and then followed the results on x-ray pictures. His results as to the cure of rickets with this method are quite satisfactory. Improvement begins between twenty-four and forty-eight hours, and it is completed in a comparatively short time. The large amount of ergosterol given did not produce any toxic effects.

ZENTAY, St. Louis.

MILK FERMENTS BEFORE PARTURITION. SHINICHI TAKAISHI, J. Pediat. (Tokyo), May 20, 1929, no. 348, p. 813.

By studying the ferment content of both colostrum and permanent milk, the author found that in colostrum before parturition the value of D is high as in colostrum just after parturition, as compared with that in permanent milk. The D value represents the amount of diastase, as measured by the method of Wohlgemuth. The same is true of the P value (P represents the index number of peptone-splitting power) which is high in colostrum and low in permanent milk. The lipase is low in colostrum both before and after parturition, and its quantity increases until the sixth to the eighth day after parturition, when it becomes fixed. The quantitative relations of these three ferments differ in every case, especially the amount of lipase, which is sometimes found in large quantities, even in colostrum, both before and after parturition. In each person, however, there is a remarkable constancy of quantitative relations in the ferments. In other words, the act of childbirth seems to exert no influence on the amount of ferments. Thus, it seems apparent that the milk ferments have no direct relation to colostrum corpuscles.

KATO, Los Angeles.

PATHOLOGY OF INFANTILE MARASMUS. JUNZO ITO, J. Pediat. (Tokyo), June 20, 1929, no. 349, p. 999.

The author performed autopsy on more than fifty patients dying of marasmus in Izumibashi Charity Hospital, Tokyo, and made a microscopic study of various organs in thirty-seven cases, which forms the basis of this paper. His general observations are: 1. There is a generalized vacuolated change in the smooth muscle cells of practically all internal organs, as well as similar changes in the vascular walls and skin. These changes are observable only in localized form in bacteremia, acute pneumonia and other forms of acute diseases. Even in chronic diseases, such as chronic indigestion and congenital syphilis, such changes are

only local. 2. In those areas of vacuolization, only the coarse fibers are seen, which may even be hypertrophied. 3. There is a morphologic and numerical degeneration of the Auerbach plexus. 4. In the sympathetic ganglions in this disease, there is a structural defect in nerve cells, but there is also a definite atrophy of the cells, together with an increase in interstitial substances. 5. There is a generalized atrophy of the lymphatic system, and the lymphoid follicles in the spleen show in part hyaline degeneration. 6. There is a marked deficiency of glycogen and of fatty substances. 7. The chromophil reaction of the suprarenal glands is noticeably decreased. 8. In marasmus complicated with intoxication the appearance of hemosiderin in the liver and spleen is marked, and there is a deposition of fat in the liver and the kidneys. 9. The most pronounced deviation from the normal is the loss in body weight rather than in height, as compared with other forms of chronic diseases.

KATO, Los Angeles.

Diseases of the New-Born

SEASONAL VARIATION IN THE WEIGHT LOSS OF NEW-BORNS. HARRY BAKWIN and RUTH M. BAKWIN, Am. J. Obst. & Gynec. **18**:863 (Dec.) 1929.

In an analysis of over 1,500 weight charts of new-born infants the authors found that the loss of weight during the first few days of life is most pronounced during the winter months. The smallest average loss was noticed between June and October, or about 15 per cent less than during the colder months.

The incidence of fever corresponded closely to the seasonal loss in weight.

The authors could not determine a relationship between the loss of weight and the nursery temperature, humidity or wet bulb depression.

ALLEN, Chicago.

TERATOMA OF THE NECK. O. SAPHIR, Am. J. Path. **5**:313, 1929.

The literature on teratoma of the neck is reviewed and a new case reported. This is a case of a teratoma of the neck in the region of the thyroid gland in a stillborn colored girl. The main constituent of this tumor was brain tissue but derivatives of the three germinal layers were present. For the first time, groups of cells resembling blood islands are described in this tumor. Even though the location suggests the thyroid gland as the origin, it is more likely that the tumor arose in the area around this gland. The tumor is classified as an embryoid teratoma according to Budde, and as a cutaneous teratoma according to Askanazy.

AUTHOR'S SUMMARY [ARCH. PATH.].

THE HYGIENE OF THE NEWLY-BORN. MORRIS GLEICH, Arch. Pediat. **46**:462, (July) 1929.

Due in no small measure to the cooperation between the obstetrician and the pediatrician there is an appreciable decrease in the morbidity and mortality of the newly born infant.

The obstetrician cares for the mother and the unborn child. The general and immediate care of the newly born infant by the pediatrician is urged, with a view to safeguarding the infant during the first precarious weeks of its existence.

WILSON, Norfolk, Va.

GENERALIZED EDEMA IN ASSOCIATION WITH TETANY OF THE NEW-BORN. W. RAY SHANNON, Arch. Pediat. **46**:549 (Sept.) 1929.

The author protests against the use of so broad a term as "edema neonatorum" to signify such a limited disease complex as generalized edema in the new-born, with high mortality.

Three cases of generalized edema associated with tetany in the new-born are reported. All the infants recovered on the administration of calcium chloride or calcium chloride and parathyroid extract-Collip.

Salt metabolism is a weak point in infants. It is probably a great factor in the fluctuations in weight seen in infancy. The sodium salts are those mostly concerned in the production of edema. Calcium salts inhibit the tendency to dropsical conditions. Calcium deficiency may also result in tetany, and hence may be the cause of edema in the new-born associated with tetany. The author claims that tetany in the new-born is common enough to indicate that calcium deficiency operates often.

Cerebral edema may be responsible for certain cyanotic attacks observed during the new-born period.

WILSON, Norfolk, Va.

STILL-BIRTH AND NEONATAL DEATH. E. CHALMERS FAHMY and ELSIE V. CROWE, Edinburgh M. J. (Tr. Obst. Soc.) **36**:165 (Sept.) 1929.

This paper gives the results of a study in which special reference was paid to the occurrence of intracranial hemorrhage and asphyxia. Reports and discussions by Holland came from the obstetric society in 1920 and from Browne in 1921 and 1924. The present communication therefore takes up the subject where the previous writers left it five years ago. It comprises a series of 300 cases; 248 full term and 52 premature infants.

The term "neonatal death" includes only infants who lived for at least an hour. The causes of death are considered in their relation to the method of delivery. Fetuses at or near full term are separated from those born prematurely. The figures presented show that the stillbirths are present in about the same frequency whether delivery is spontaneous (vertex), by forceps or by breech presentation, but the neonatal deaths are highest following spontaneous delivery in vertex presentations. The causes of death in the full term infant are analyzed and discussed. In forty-seven stillbirths, eleven were due to intracranial hemorrhage and eight to hemorrhage plus asphyxia.

Among the sixteen cases of full term neonatal deaths, seven were due to intracranial hemorrhage, three of which were without dural tear, three with injury of the tentorium and one with injury of the superior longitudinal sinus. All the infants showed asphyxia at birth. All were born with easy labor ranging from three and one-half to eighteen hours.

The effect of forceps delivery, toxemia, breech delivery, version, cesarean section and prematurity as well as the consideration of craniotomy are discussed at some length.

The authors emphasize in conclusion the necessity for putting to clinical use the knowledge that intracranial hemorrhage and asphyxia may occur together or separately. They urge the utmost gentleness in the handling of the infant born in asphyxiated conditions. Asphyxia pallida is probably a condition of shock.

NEFF, Kansas City, Mo.

DEATH AT BIRTH FROM HEMORRHAGE INTO THE THYROID GLAND. MUTEL and MORIN, Ann. de méd. lég. **8**:157, 1928.

A woman, aged 28, the mother of two children, with whose birth there was no difficulty, found her third child dead about twenty-four hours after birth. This child also was born in normal labor and apparently was well when born. Because of anonymous accusations, a postmortem examination was made, and the only alteration found anywhere was a hemorrhage into the thyroid gland. The gland was twice the normal weight, the hemorrhage being entirely within the capsule, and in the other structures of the neck there was no evidence of injury.

It is well known that spontaneous hemorrhages into the suprarenal glands may cause death in both infants and adults. About seven deaths from hemorrhage into the parathyroid glands in infants from 3 to 6 months old have been reported.

Similar hemorrhages into the hypophysis with corresponding serious consequences apparently have not been observed. Goiterous swelling of the thyroid gland due to hyperemia caused by passage through the birth canal is well known, and usually yields quickly to hot packs and other local treatment. Hemorrhages such as this reported by Mutel and Morin causing death are extremely rare. It was difficult to find any thyroid tissue microscopically, because of its mechanical displacement and destruction by the apoplexy in the gland.

[ARCH. PATH.]

THE DIGESTIVE CANAL OF THE NEW-BORN. G. ALBANO, Monatschr. f. Kinderh. **45**:306 (Nov.) 1929.

In a series of studies with the x-rays, the writer has investigated the anatomy and physiology of the gastro-intestinal tract of the new-born.

The stomach is usually pear-shaped, pointing downward. It is usually vertical and lies to the left of the spinal column. The duodenum is straight and crosses the midline from above downward and from left to right. The last coil of the small intestine, in contrast to that in adults, crosses over and often descends sharply in the direction of the cecum, which lies above the right iliac crest. The colon in all its divisions is short.

In its motility and emptying time the stomach is variable. It is possible that the bismuth clings to the mucous membrane and is not all emptied into the intestine for some days. Thus one can readily be mistaken in attempting to draw diagnostic conclusions. Such a process does not in any way interfere with the digestion of the breast milk. The child shows absolutely no disturbance.

The distal portion of the appendix is filled in 10 per cent of cases. The writer believes that diagnosis by means of x-rays in the new-born is of doubtful value physiologically though anatomic relations are shown.

GERSTLEY, Chicago.

THE CHARACTER AND DURATION OF THE POSTPARTUM BLOODSTREAM IN THE UMBILICAL VESSELS. G. HASELHORST, Ztschr. f. Geburtsh. u. Gynäk. **96**: 487, 1929.

The increase in weight from the umbilical blood of newly born infants averaging 3,440 Gm. was 114 Gm. The time for the increase varies between two and twenty minutes and may last over thirty minutes in individual cases.

Transitory weight loss may be observed but only in cases in which the umbilical arteries pulsate for a long time.

The vascular system of the infant, including the placenta and vascular organs, possesses the ability to adjust itself in a remarkable manner to changes in the blood volume.

It is probable that, beside the regular circulation between fetus and placenta, movement of blood masses participates in the chemical interchange.

ADAIR, Chicago.

THE WEIGHT PROPORTION BETWEEN AFTER-BIRTH AND CHILD: IS THE PROPORTION OF ANY VALUE FOR THE DIAGNOSIS OF CONGENITAL SYPHILIS? TORVALD BRANDT and KAREN SIECK, Acta obst. et gynec. Scandinav. **8**:431, 1929.

The authors give a statistical analysis of 4,000 normal and 396 syphilitic cases. Their observations confirm those of Westermark (Acta obst. et gynec. Scandinav., 1925, vol. 4) that large children have large, and small children have small, after-births, that premature infants have relatively larger placentas and that the ratio is different in the different sexes.

The average weight of the newly born infants was 3,497 Gm., and of their placentas, 666 Gm.; there is a definite weight ratio (from 1:3.0 to 1:7.6) but 3 in 1,000 fall outside this proportion.

Of 396 syphilitic infants, 134 were never treated. The weight ratio of placenta and fetus does not vary markedly from the normal. The proportions are the usual ones for macerated fetuses. One cannot draw any conclusions as to the existence of syphilis from the relative weight of the placenta, as it is not different in syphilitic and nonsyphilitic infants.

A relatively heavy placenta is a sign of prematurity and not of syphilis. Pinard's law that syphilis can be diagnosed from a heavy placenta is not tenable and should be discarded.

ADAIR, Chicago.

McCLURE-ALDRICH SKIN REACTION IN NEWLY BORN BABIES. I. PETERI, Orvosi hetil. **73**:1240 (Dec. 14) 1929.

The McClure-Aldrich method of injection of 0.8 per cent saline solution in the amount of 0.2 cc. intracutaneously was used in twenty-three newly born babies. Every infant received two injections, the first on the day of birth and the second four or five days later. This technic was used to study the behavior of absorption at a time when the newly born infant has a rich supply of water in its tissues and while it is supposedly poorest in water supply of the body. The function of the injected saline in newly born infants seems to be quicker than in older infants as given in the literature. It disappeared in about twenty-seven minutes on the first day of life and in about thirty-two minutes on the fifth day. This would mean that in the stage of exsiccosis, the injected saline would be absorbed somewhat more slowly than in the stage of good water supply. In some of the infants a third injection was given on the eighth day of life when the time absorption was about the same as on the first day.

ZENTAY, St. Louis.

BIRTH INJURIES IN THE NEW-BORN. II. CLINICAL OBSERVATIONS OF INTRACRANIAL HEMORRHAGE IN NEW-BORN INFANTS. HIDEO YAGI, Jap. J. Obst. & Gynec. **12**:130 (June) 1929.

The author made a previous report in 1927, in which he stated that intracranial hemorrhage was the most frequent cause of fetal death during labor and in the first puerperal week. During three years there were 3,671 labor cases, and excluding fetuses under seven months' gestation, there were 308 stillbirths and deaths during the first week.

Autopsy was performed in 178 cases. Intracranial hemorrhage was found in 62 of these, or in 34.3 per cent.

The author made clinical observations on forty infants, thirty-three of whom died.

The child may be born deeply asphyxiated and be difficult to resuscitate; some show twitching or opisthotonus, others have no signs except deep asphyxia.

The author classifies the cases as follows:

1. Excited motility, or "erethic form," with cyanosis, restlessness, vomiting, difficulty in swallowing, irregular respiration, tense and pulsating fontanel, twitching of the face or extremities, general convulsions, and perhaps opisthotonus and rigidity of the neck.

2. Paralyzed motility, or "torpid form," with cyanosis, inability to nurse, feeble pulse, hypothermia and sleepiness. These patients usually die in two or three days. Those with the "erethic form" may recover or may pass into the "torpid form."

The author investigated 2,125 cases in the Infants' Clinic and found sixty-nine possible cases, or 3.5 per cent, of the total observed. From an analysis of these cases with reference to the labor, the author concluded that from 29 to 42 per cent gave evidence of intracranial hemorrhage which presumably had a close connection with the mental and physical development. In 29 per cent there was a history of operative delivery and asphyxia, and in 42 per cent of dystonia.

ADAIR, Chicago.

BIRTH INJURIES IN THE NEW-BORN: V. DIAGNOSIS OF INTRACRANIAL HEMORRHAGE IN NEW-BORN CHILDREN BY ROENTGEN RAYS. HIDEO YAGI, Jap. J. Obst. & Gynec. **12**:223 (Sept.) 1929.

The author calls attention to the difficulty in obtaining autopsies on fetuses and newly born infants and suggests the use of the tray as a means of making the diagnosis of intracranial hemorrhage.

The method was mentioned by L. Shoenholz (*Klin. Wchnschr.* **5**:1775, 1926), and Pischzek (*Zentralbl. f. Gynäk* **51**:2635, 1927), used a fluid mixture of 40 per cent sodium bromide for injection.

The author followed the foregoing procedure by injecting the following solution into the internal jugular vein: red oxide of lead, 60; gelatin, 20; chalk, 20, and distilled water, 100. He also utilized this solution satisfactorily without the chalk. It is fluid at 40 C., but becomes solid at a lower temperature. Gradual and careful injections are necessary to avoid artificial rupture of the vessels. The amount injected varies from 60 to 100 cc., with an average of 80 cc. Avoid forced pressure and keep the body immersed in warm water.

The author also used Pischzek's method of injecting 40 per cent sodium bromide solution; it is not necessary to keep the body warm. This method does not give as satisfactory x-ray pictures as that of Shoenholz.

He experimented with other substances and found barium sulphate (40 Gm. to 100 cc. of water) to be satisfactory.

He examined about thirty cases, in some of which the observations were corroborated at autopsy. The roentgenograms, especially the stereoscopic pictures, are valuable in diagnosing cases of intracranial hemorrhage, as well as in studying the vascular supply of the brain: 1. Subdural hemorrhages are shown by the presence of irregular shadows of varying sizes. 2. Ventricular shadows are also shown. 3. It is difficult to show the difference between subdural and intracerebral hemorrhages. 4. No definite differences between leptomeningeal and subdural hemorrhages are shown.

In summarizing, the author calls attention to the danger of confusing suffocation and asphyxia apoplectica. He states that the roentgen examination is the only means of verifying the presence of intracranial hemorrhage except by autopsy. He feels that the method of lumbar puncture is not reliable for diagnosis.

The best solutions are red oxide of lead and barium sulphate, the latter being more convenient and as reliable to inject.

ADAIR, Chicago.

Acute Infections

INFECTIONS PROBABLY DUE TO MORGAN'S BACILLUS. RIGNEY D'AUNOY, Am. J. M. Sc. **178**:834 (Dec.) 1929.

The author discusses the morphology, cultural reactions, pathogenicity and serologic interrelationship of Morgan's bacillus and gives three case reports in which he was able to isolate the bacillus.

The predominating symptom in the first case was pyrexia, in the second, pyelitis and in the third, colitis. One strain isolated proved pathogenic for small laboratory animals. Agglutinins were demonstrated for homologous organisms in the blood of all three patients.

HENSKE, Omaha.

POOLED ADULT BLOOD SERUM AS A PROPHYLACTIC MEASURE IN A MEASLES EPIDEMIC IN AN INSTITUTION. ALVIN E. SIEGEL and HORTENSE ERMANN, Am. J. M. Sc. **179**:192 (Feb.) 1930.

During an epidemic of measles in Philadelphia, in which a number of the children living at the Southern Home for Destitute Children were exposed to measles, the author decided to protect the susceptible children against measles. Of the 100 children in the home, 45 had had measles in the home or gave a history of measles prior to admission, and 55 gave no history of having had measles. As the serum of convalescent patients was not available in sufficient

quantities, it was decided to use pooled blood from adults who had had measles earlier in life; the 55 susceptible children were inoculated with 5 cc. of the pooled serum by injection into the buttocks. None of the children receiving the serum developed measles or had attenuated or modified measles.

HENSKE, Omaha.

WHOOPING COUGH: THE SITE OF THE LESION. LOUIS W. SAUER and LEONORA HAMBRECHT, Arch. Path. **8**:944 (Dec.) 1929.

Autopsy material was studied in three cases of whooping cough: one a ringtail monkey which was killed when beginning to recover from an uncomplicated case of experimental pertussis, one an infant, aged 3 months, who died twelve days after the beginning of the cough, with convulsions and bronchopneumonia, and one a *Maccacus rheusus* monkey which was killed at the beginning of bronchopneumonia complicating experimental pertussis. In all cases the larynx and trachea showed no injury. The most profound changes in the uncomplicated case were in the region of the smaller bronchi and larger bronchioles; these were filled partially with desquamated epithelium, lymphocytes, plasma cells and a few polymorphonuclear leukocytes. The epithelium and submucosa showed definite round-cell infiltration which was sufficient in places to obliterate the lumen (endobronchiolitis obliterans). When there was beginning bronchopneumonia the smaller bronchioles were involved and pertussis bacilli were found even in the alveoli.

LESLIE, Evanston, Ill.

THE EARLY DIAGNOSIS OF PERTUSSIS. SAMUEL Z. ORGEL, Arch. Pediat. **46**: 149 (March) 1929.

Intracutaneous injections of freshly made *Bacillus pertussis* vaccine, 2,000 million per cubic centimeter, less than a week old, were given. Control injections of a nonspecific milk preparation, colon bacillus vaccine (400 million bacilli per cubic centimeter) and a vaccine containing streptococci and staphylococci (100 million of each, per cubic centimeter) were employed. The tests were used on 1,094 children, of whom 245 had pertussis, 330 were suspected of having it and 190 had previously had it. Tables are published showing in detail the results of the tests. The author concluded that a positive intradermal skin reaction obtained by the use of bacillus pertussis vaccine, containing 2,000 million bacilli per cubic centimeter, is not diagnostic of pertussis.

WILSON, Norfolk, Va.

INFLUENZA: FORMS AND SYMPTOMATOLOGY; EPIDEMIC 1928-1929. JOSEPH C. REGAN, Arch. Pediat. **46**:201 (April) 1929.

In the recent epidemic, the disease may be classified as follows: (1) generalized, (2) gastro-intestinal and (3) respiratory types, and (4) manifestations of the nervous system. The epidemic appeared to differ from the epidemic of 1918-1919 particularly in that it was milder, the extremely severe hemorrhagic forms of bronchopulmonary complications were rare, the influence of the disease on pregnancy and the pregnant woman was less conspicuous and there appeared to be a high incidence of the gastro-intestinal type of case, especially in children. There is a detailed description of the protean manifestations of influenza as it occurred in children during the epidemic.

WILSON, Norfolk, Va.

PYELITIS IN CHILDHOOD: A STUDY OF HOSPITAL RECORDS. ROY P. FORBES, Arch. Pediat. **46**:245 (April) 1929.

The incidence of pyelitis in 16,367 admissions to the Children's Hospital of Denver was 0.53 per cent. A moderate pyuria is frequently observed during infections of the throat. The finding of pus cells during a febrile attack does not justify a diagnosis of pyelitis. In the eighty-seven cases reported, twenty-nine complicated

respiratory infection and in most of these the infection was in the upper respiratory tract; 94 per cent occurred in girls. There were no deaths except when there were concurrent infections (in four cases: nephritis, otitis media, pneumonia and paratyphoid fever). "Bacteriologic cure" with sterile urine on two occasions is suggested.

WILSON, Norfolk, Va.

SERUM TREATMENT IN CEREBROSPINAL MENINGITIS. EDWARD L. BENJAMIN, Arch. Pediat. **46**:252 (April) 1929.

Nine cases are reported. There was no history of exposure. Intensive early treatment, intraspinally, combined with intravenous and intramuscular treatment gives the best results. Seven of the nine patients recovered. With the appearance of the reduction of sugar improvement followed. Cisternal puncture should be resorted to more often in dry taps since it is attended by no great danger to the patient when properly done. Prognosis is influenced by early recognition and early treatment.

WILSON, Norfolk, Va.

THE NERVOUS COMPLICATIONS OF MEASLES. PAUL K. JENKINS, Arch. Pediat. **46**:257 (April) 1929.

A girl, aged 7 years, with measles developed evidences of meningitis and cerebral irritation which continued for six days. She then made an uneventful recovery. In such conditions the prognosis as to life is good, the mortality being about 10 per cent. About 65 per cent of the patients who survive have residual symptoms.

WILSON, Norfolk, Va.

DIPHTHERIA: REPORT OF A CASE FOLLOWING TOXIN ANTITOXIN ADMINISTRATION AND NEGATIVE SCHICK TEST. EDWARD LEWIS, Arch. Pediat. **46**:260 (April) 1929.

A girl, aged 6 years, who had been given toxin-antitoxin three and one-half years before, developed diphtheria. The Schick test gave negative results six months before the attack and three years after the inoculation; 10,000 units of antitoxin were given and there was a marked serum reaction. Immunity must have disappeared within approximately six months after the Schick test or within three and one-half years after active immunization. The history of previous active immunization against diphtheria, even though confirmed by a rather recent negative Schick reaction, should not alone preclude diphtheria as a diagnosis, nor prevent the institution of early and adequate antidiphtheritic treatment in suspected cases.

WILSON, Norfolk, Va.

A SECOND ATTACK OF SCARLET FEVER WITHIN EIGHTEEN MONTHS. WINGATE M. JOHNSON, Arch. Pediat. **46**:262 (April) 1929.

A girl, aged 7 years had scarlet fever and eighteen months later developed classic scarlet fever which was more severe than the first attack.

WILSON, Norfolk, Va.

MALIGNANT DIPHTHERIA. PHILIP S. POTTER, Arch. Pediat. **46**:430 (July) 1929.

The symptoms are discussed, and two cases are reported, in which the patients died. The circumstances connected with the cases were unusual in that the children played in a stream which communicated with a sewer. Immunized members of the family, with the exception of the father, gave positive cultures. A visiting aunt who refused immunization died with diphtheria. There is a discussion of the possibility of infection from the open sewer.

A few others in the community gave positive cultures, and these were not contacts of the patients.

WILSON, Norfolk, Va.

DIPHTHERIA FATALITIES WITH SPECIAL REFERENCE TO CIRCULATORY FAILURE. JOSEPH GREENGARD, Arch. Pediat. **46**:411 (July) 1929.

In an attempt to determine the cause or causes of fatalities, 116 deaths occurring in 726 cases of diphtheria admitted to the Municipal Contagious Disease Hospital of Chicago were studied; 82.7 per cent of the deaths occurred in early childhood; 97.3 per cent of the patients who died were white, 3 were colored and 1 a Japanese. Of 457 deaths from diphtheria in Chicago in 1928, 5.2 per cent were in the colored race (23 negroes and 1 Japanese). During an eight year period, 4.5 per cent of the deaths were in the colored race. This is less than the percentage of the colored population (5 per cent). A difference in racial susceptibility is suggested. The total mortality was 16 per cent. Circulatory failure was the principal cause of death, accounting for 80 per cent of the fatalities. Neglect in the administration of antitoxin seems to be the most important factor in these fatalities. It is possible that certain epidemics are characterized by a highly virulent strain of organism which overwhelms the host rapidly.

WILSON, Norfolk, Va.

AN EPIDEMIC OF ERYTHEMA INFECTIOSUM, "THE FIFTH DISEASE." EUGENE H. SMITH, Arch. Pediat. **46**:456 (July) 1929.

Three epidemics have been reported in the American literature. The disease is only mildly contagious, and subjective symptoms are slight and may be absent though the rash be marked and extensive. The eruption first appears on the cheeks as fine red papules or macules, which soon become confluent. On the limbs it develops first on the outer aspect of the arms and forearms, on the thighs and especially on the buttocks. Later the flexor surfaces of the upper extremities are involved, and the condition may extend as far as the hands and fingers. A characteristic feature is the peculiar gyrate, circinate or maplike form of the lesions; especially is this noticed on the flexor surface of the forearms and the buttocks. The color of the eruption in these regions, when fully developed, is usually dusky red or bluish. It may be slightly elevated or urticarial. On the body it is much more scattered, smaller, papular and macular, and the irregular forms seldom occur. The duration of the disease is from a few days to three weeks, during which time the rash may almost completely fade and reappear several times. The disease does not appear to be highly contagious. During the fall of 1927 and spring of 1928, fourteen cases came to the attention of the author.

WILSON, Norfolk, Va.

RESEARCH IN CONNECTION WITH ACUTE POLIOMYELITIS. A. E. VIPOND, Arch. Pediat. **46**:479 (Aug.) 1929.

Eight cases are reported. A streptococcus vaccine secured from the urine of patients with poliomyelitis was employed in treatment. Three patients who were treated during the first three days recovered completely. Two who were treated on or before the tenth day improved. The author believes that the vaccine, prepared as described, is effective if begun early.

WILSON, Norfolk, Va.

SEVERE HEMOGLOBINURIA IN A CHILD, OCCURRING IN THE PRODROMAL STAGE OF CHICKENPOX: REPORT OF CASE. ALBERT D. KAISER and WILLIAM L. BRADFORD, Arch. Pediat. **46**:571 (Sept.) 1929.

A case is reported in a boy aged 2½ years, of Jewish parentage. About six days before lesions of chickenpox appeared he had hemoglobinuria associated with

what was thought to be mild sore throat. This resulted in a severe secondary anemia, with 1,000,000 red cells per cubic millimeter and hemoglobin (Sahli) 15 per cent; the white cell count was 61,300. After several transfusions of blood from several donors he was discharged in good condition about seventeen days after the onset of the condition.

The various types of hemoglobinuria are discussed.

In the case reported the two most probable causes for the extreme hemolysis were paroxysmal hemoglobinuria and some type of acute infection. Physical examination disclosed no disease of the throat or tonsils. There was a positive Donath-Landsteiner reaction obtained by chilling the serum during the height of the attack of hemoglobinuria; since it could not be demonstrated after the attack, it was obviously not of the paroxysmal type.

WILSON, Norfolk, Va.

THROMBOSIS OF EXTERNAL PUDIC ARTERY COMPLICATING CHICKENPOX. ADDISON H. BISSELL, Arch. Pediat. **46**:588 (Sept.) 1929.

A case is reported in a girl, aged 8 years. The onset occurred on the fifth day of a mild attack of chickenpox. The temperature rose from normal to 106 F. in eight hours. The patient complained bitterly of abdominal pains in the left side just above the inguinal region, and in the upper and inner part of the thigh. The skin in this area was dark red, with swelling in the inguinal area and with early necrosis in the center of the inguinal swelling. Daily injections of 500,000 units of erysipelas antitoxin were given for three days, though the lesions did not have the typical appearance of erysipelas. There was daily improvement but after forty-eight hours a culture showed *Staphylococcus aureus*. The cellulitis extended up to the costal margin, laterally to the posterior axillary lines and for 3 inches (7.62 cm.) down both thighs. On the ninth day a well demarcated area of gangrene was excised with exposure of the abdominal muscles in this region. Thrombosed vessels were seen under the lower edge of the cavity; these were thought to be the superficial external pudic artery and vein. Pain, swelling, tenderness and discoloration disappeared after about five weeks.

WILSON, Norfolk, Va.

COMBATING CONGENITAL SYPHILIS. L. B. DICKEY and T. L. SUTTON, California & West. Med. **31**:242 (Oct.) 1929.

The incidence of congenital syphilis in this series was about 2 per cent for a clinic practice. Early and intensive treatment is advocated. A combination of sulpharsphenamine, bismuth and mercury, in relatively large doses given twice weekly offers a rapid serologic cure in practically all infants. The disease yields to similar therapy in a fair proportion of older children. The percentage of cures in this group would be increased by intensive work in social service. The mortality in this series was about 10 per cent (ten in 103 patients treated). The immediate cause of death was not related to the treatment, except possibly in one case. Many infants with a history of syphilis in the mother, and with a positive Wassermann reaction of the blood present no signs. These patients should be treated the same as if there were obvious lesions.

DICKEY, San Francisco.

MENINGOCOCCUS INFECTION: ITS TREATMENT. EDWARD B. SHAW and H. E. THELANDER, California & West. Med. **31**:309 (Nov.) 1929.

Shaw and Thelander report three cases of epidemic meningococcus meningitis occurring in the same family. All three patients had meningococci in the throat. The authors stress the importance of purpuric manifestations in early diagnosis, and have diagnosed cases early by finding meningococci in stained smears from venous blood. They advocate both intraspinal and intravenous treatment in early

cases, and intraspinal treatment in later cases. The dose of the antiserum should be large. Intraspinal treatments should be repeated at eight or twelve hour intervals, and with improvement should be repeatedly daily. Treatment should be stopped only when the organisms have disappeared from the spinal fluid. If block occurs so that intraspinal administration is impossible, the serum should be injected into the cisterna or the ventricles. DICKEY, San Francisco.

EPIDEMIC CEREBROSPINAL MENINGITIS. B. E. BONAR, California & West. Med. **31**:316 (Nov.) 1929.

Bonar reports the observations on seventy-one cases of cerebrospinal meningitis seen in the Salt Lake General Hospital. Various complications occurred, but sequelae of the paralytic type were uncommon. The general treatment is described, together with specific therapy. The mortality was about 50 per cent in this series.

DICKEY, San Francisco.

CONVALESCENT SERUM IN THE PREVENTION OF MEASLES. W. WARWICK, Canad. M. A. J. **21**:694 (Dec.) 1929.

The serum of convalescent patients was used in an institution in which sixty-seven children had been definitely exposed to the measles; 10 cc. was given about four or five days after exposure. About ten of the children developed slight atypical signs but none had severe attacks of measles. A similar result was obtained in ten children exposed in the children's ward of the General Public Hospital. The author urges the use of the serum especially in institutions or when infants or debilitated children have been exposed. MOORE, Omaha.

PYELITIS IN INFANCY. DOUGLAS D. MARTIN, J. Florida M. A. **16**:256 (Dec.) 1929.

In a series of seventy-five cases of pyelitis treated in the past two years there was but one male child. The ages have been from 2 months to 11 years. In relatively severe cases the leukocyte count was nearly normal with a normal polymorphonuclear count. The count was proportionally higher in mild cases than in severe cases. Congenital anomalies probably account for prolonged duration of cases that have not responded to usual treatment. Pyelitis is often confused with appendicitis and pneumonia. In cases that do not yield to commonly accepted treatment, daily irrigation of the bladder with boric acid solution and the intravenous injection of methenamine were found useful. Vaccines have not proved of benefit.

LOVE, Jacksonville, Fla.

SKIN REACTIONS TO FILTRATES AND KILLED CULTURES OF GREEN-PRODUCING COCCI IN RELATION TO MEASLES. WILLIAM L. BRADFORD, J. Infect. Dis. **44**:378, 1929.

No definite relationship was found to exist between the immune and non-immune to measles as determined by cutaneous reactions to the toxic filtrates and antigens of certain green-producing cocci, including the Tunnicliff coccus, the organism of Duval and Hibbard and *Streptococcus morbilli* of Ferry.

AUTHOR'S SUMMARY [ARCH. PATH.]

HYPERSENSITIVENESS TO DIPHTHERIA BACILLI. JAMES M. NEILL and WILLIAM F. FLEMING, J. Infect. Dis. **44**:397, 1929.

The hypersensitiveness described agreed with the previous example of diphtheria hypersensitiveness in two respects: There were immediate skin reactions to either diphtheria or xerosis diphtheroid material, and the hypersensitive reactivity could

be transferred to local skin areas of normal persons. The two states of hypersensitiveness were not identical, however. The first distinction was that the ratio of diphtheria reactivity to xerosis reactivity was different for the two persons; one being more reactive with the xerosis diphtheroid than with diphtheria, and the other being more reactive to diphtheria than to the diphtheroid. The second distinction was that the one was reactive with a heat stable constituent of the diphtheroid filtrate while the diphtheroid constituent to which the other person reacted was heat labile. Although both persons gave the same type of skin reaction to both the diphtheria and the diphtheroid bacteria, the described distinctions suggest a difference in the bacterial origin of their sensitization.

AUTHOR'S SUMMARY [ARCH. PATH.]

THE SCHICK TEST IN PALESTINE, A COUNTRY OF LOW DIPHTHERIA PREVALENCE. A. MANN and I. J. KLIGLER, J. Prev. Med. **3**:309, 1929.

Diphtheria and scarlet fever are relatively far less prevalent in Palestine than in countries having a temperate climate. From about 3,000 Schick tests it appears that the percentage of diphtheria immunes at the ages above 8 in Palestine is the same as found by Zingher in New York City, but that the immunization starts earlier among the Palestine children than in the New York children. A comparison of the native and foreign born groups on the one hand, and the Ashkenazic and Sephardic communities on the other, indicates that the native born, especially in the Sephardic community, develop their diphtheria immunity much earlier than the foreign born children. It would seem, therefore, that the absence of diphtheria in Palestine is only apparent. Since active immunity can probably be acquired only as the result of infection, the infection rate in the early age groups must be relatively high to produce so large a number of immunes.

AUTHORS' SUMMARY [ARCH. PATH.]

CASE OF UNDULANT FEVER IN AN INFANT. CARL O. KOHLBRY, Minnesota Med. **12**:414 (July) 1929.

This is the report of a case of undulant fever in a male infant, aged 1 year, with recovery. The disease is rare in children under 4 years of age.

HUENEKENS, Minneapolis.

AN ARGUMENT IN FAVOR OF VACCINATION, WITH STATISTICS OF THE INCIDENCE OF SMALLPOX IN THE UNITED STATES, ITS DEPENDENCIES AND CANADA. SAMUEL B. WOODWARD, New England J. Med. **202**:122 (Jan. 16) 1930.

The author presents the smallpox situation in the United States from 1913 to 1923, with a total of 579,610 reported cases, and tables of the cases in the respective states, its dependencies and Canada from 1919 to 1928, totaling 589,076. These figures, however, do not constitute the actual number, since a large percentage are not reported to the authorities. In the United States today smallpox is more prevalent than in any country in the world, with the possible exception of China, India and Russia. Cases are few in the dependencies as compared with those in the United States.

Vaccination laws are most nearly enforced in the Eastern states, but not rigidly enough even there. Parochial and private schools and religious sects furnish a large percentage of the cases on account of the lack of compulsory vaccination. In spite of the many cases, health officers in many sections do not seem to sense the necessity for rigid enforcement of the vaccination laws. The author asks the aid of the physicians in strengthening the laws protecting against smallpox.

GENGENBACH, Denver.

TYPHOID-PARATYPHOID VACCINATION IN CHILDHOOD. HESSE, Arch. de méd. d. enf. **32**:729 (Dec.) 1929.

The value of antityphoid vaccination was so well attested during the late war that compulsory administration of this specific agent is observed in practically all European armies. The result of this almost universal prophylaxis among young men is noted in the comprehensive review of Hesse, who finds that the number of female patients admitted to the typhoid wards of the general hospitals in Paris is now five times greater than the number of males. This is the exact reverse of prewar conditions. With many children augmenting the figures for adults, the question of obligatory vaccination in early life has been raised by Chauffard and Vincent, who contend that it has become as important a preventive measure as vaccination against smallpox. It appears that these injections may be given to children without the reaction so frequently noted in adults.

Typhoid is rare in patients under 2 years of age, but Renaud and Duchein recommend inoculation even at this period in consideration of its benign character. After the second year the incidence of typhoid mounts rapidly; statistics from Alsace-Loraine show that 30 per cent of all cases occur before the age of 15. In discussing the two modes of vaccination, the subcutaneous and the buccal route, the former method is recommended, although the experience of Besredka would seem to show a fair degree of protection from oral administration. There are four varieties of polyvalent vaccines now employed subcutaneously, each of these immunizing against infections by the Eberth bacillus and the organisms of paratyphoid A and paratyphoid B.

In vaccination of children by the oral route, two products are now in use, the enterovaccine of Lumière and the bilivaccine of Besredka. Hesse advises the simultaneous inoculation of one of these specifics with toxin antitoxin or anatoxin in early infancy, and every two years thereafter, with typhoid vaccine alone.

AMESSE, Denver.

TREATMENT FOR WHOOPING COUGH BY VACCINE. K. KYRIAZIDÈS, Paris méd. **2**:473 (Nov. 30) 1929.

Autogenous vaccine from the Bordet-Gengou bacillus was made, and injections of 25, 50, 100, 250 and 500 millions were given. In the epidemic in Athens in 1925 this was used in 113 cases with suitable controls. The results were favorable, the disease running a very short course and the catarrhal symptoms being partly decreased.

SANFORD, Chicago.

THE NECESSITY OF TAKING CULTURES FROM THE THROATS OF SCARLET FEVER PATIENTS. F. COSTE, M. LEBLOND and P. E. VANNIER, Presse méd. **37**:1405 (Oct. 30) 1929.

The authors emphasize the necessity of taking cultures from the throats of scarlet fever suspects. Hemolytic streptococcus found in the absence of exanthemas do not necessarily mean that the patient has the disease. On the other hand, with an exanthem the absence of organisms would make the diagnosis doubtful. This method is also of value in determining the presence or absence of contacts and carriers.

SANFORD, Chicago.

FOUR YEARS' EXPERIENCE WITH DIPHTHERIA TOXOID. M. and G. MOZER, Presse méd. **37**:1543 (Nov. 27) 1929.

The use of anatoxin was begun four years ago at the Marine Hospital at Berck. There has never been a serious accident in inoculation. In 1924 and 1925, before this method was adopted, there were 103 cases of diphtheria and 8 deaths. In 1928 and 1929, following its adoption, there were 5 cases with no complications. The authors conclude that diphtheria toxoid immunization carried out systematically has been highly successful in the Marine Hospital.

SANFORD, Chicago.

PROPHYLAXIS OF MEASLES WITH CONVALESCENT SERUM AND CAUSE OF ITS FAILURE. PIERO BRUSA, Riv. di clin. pediat. **27**:723 (Oct.) 1929.

The author reports two instances in which the serum of convalescent patients was used in an endeavor to protect exposed children from developing measles. In one instance (twenty-four children) no cases of measles developed; in the other, measles developed in the four children exposed and given serum.

The failure of the serum to protect in the latter group is ascribed by the author to the fact that the serum was obtained from three children between 14 and 18 months of age. Children in the second year of life are ordinarily poor producers of antibodies.

HIGGINS, Boston.

ADENITIS: USE OF ANTIDIPHTHERITIC SERUM. J. BONABA, M. A. JANREGUY and A. NOGUÉS, Arch. latino-am. de pediat. **23**:697 (Sept.) 1929.

The observations of the authors are based on five cases. Adenitis can be a part of the picture of serum disease. It may follow the use of any animal serum.

It generally supervenes on about the sixth to the fourteenth day after the first injection of serum, and almost always coincides with other phenomena of serum disease.

It may become extensive and may resemble acute inflammatory adenitis. The outcome is always favorable and the condition subsides after a short duration. No suppuration of the enlarged glands has ever occurred.

SCHLUTZ, Minneapolis.

EXANTHEMS OF CHILDHOOD. E. FILGUEIRAS, Arch. de pediat., Rio de Janeiro **2**:431 (Oct.) 1929.

The different forms of rash that the atypical cases of scarlet fever may assume is discussed. In many of these cases the Dick test is the only satisfactory means of diagnosis. It is necessary to diagnose these obscure cases if contagion is to be kept down.

SANFORD, Chicago.

STAPHYLOCOCCUS SEPTICEMIAS. A. CASTELLANOS, Bol. Soc. cubana de pediat. **1**:539 (Oct.) 1929.

Two clinical cases are discussed in full, with the treatments used in these conditions. Colloid therapy, protein therapy, transfusions and vaccines have not been satisfactory. Castellanos believes that 2-ethoxy-6,9-diamino-acridine hydrochloride offers the best source of treatment. This is given in the dosage of 1 mg. per kilogram of body weight, in a 1 per cent solution. The injections are given intravenously, and several may be given at twenty-four to forty-eight hour intervals. The only symptom of intolerance is a slight fever that only lasts a few hours.

SANFORD, Chicago.

TYPHOID FEVER IN A CHILD AGED FOUR YEARS. MARTÍNEZ VARGAS, Med. de los niños **30**:259 (Sept.) 1929.

This case is reported to show the result of injections of Llorente's antityphoid serum. A boy, aged 4 years, had been in contact with an adult suffering with an infectious fever, probably typhoid. Ten days later he developed fever, diarrhea and stupor. In the five days following he had two attacks of nosebleed. Rose spots and splenomegaly soon followed. Blood cultures were positive for the typhoid bacillus, the Widal test showed agglutination in dilutions of 1:80 and the benzidine test showed the presence of blood in the stools. The treatment consisted of sulphate of soda with betanaphthol benzoate, with daily irrigations with a solution containing powdered tannin and salicylic acid. He was also

given a daily subcutaneous injection of antityphoid serum Llorente for ten days. He was discharged as cured on the twenty-ninth day.

DWYER, Kansas City, Mo.

THE SPONTANEOUS CURE OF DIPHTHERIA. J. SIEGL, Arch. f. Kinderh. **88:** 154 (Aug. 20) 1929.

The author gives a summary of nineteen patients who varied in age from 8 months to 8 years; all gave positive cultures of *Bacillus diphtheriae* from the throat and nose. The highest temperature was 40 C. (104 F.), and the period of sickness varied from three to twenty-five days. These patients gave a positive reaction to the Schick test before the sickness, and a negative reaction afterward. All recovered spontaneously.

SANFORD, Chicago.

PROPHYLACTIC VACCINATION AGAINST CHICKENPOX. D. OROSZ, Arch. f. Kinderh. **88:**269 (Oct. 25) 1929.

After a study of the von Késmársky method of intracutaneous injection of 0.1 cc. of fresh blood taken from a patient during the first thirty-six hours of the eruption, Orosz expresses his belief that the variability of the cases that serve as a point of origin for the vaccination are more important than the age of the child.

SANFORD, Chicago.

CONTRIBUTIONS TO THE KNOWLEDGE OF ACTIVE IMMUNIZATION AGAINST DIPHTHERIA. R. OTTO and G. BLUMENTHAL, Deutsche med. Wchnschr. **55:** 1243 (July 26) 1929.

The research reported in this paper was instigated by the report on the results of diphtheria vaccination, in Berlin (abstr., AM. J. DIS. CHILD. **38:**1300 [Dec.] 1929). The percentage of negative reactions to the Schick test obtained after the vaccination was much lower than those reported from other countries. This was laid to the number of injections as reported both by Seligman and the essayist. The work was done with two preparations of German manufacture and an anatoxin. Guinea-pigs were used for the experimental work and were given 1 cc. of the preparation, the dose for human subjects. The results were checked by the antitoxin titer in antitoxin units according to Romer's technic.

The results, fully tabulated and explained, showed that after one injection only a slight result was obtained. This was definitely increased by a second injection with all three preparations. A still greater rise of the antitoxin titer was obtained by a third injection with two of the preparations while a third decreased the titer. After from three to five months all the animals were given five times the lethal dose of diphtheria toxin. The results showed definitely that the high titer values protected them against the toxin.

The preparations tested were all fully neutralized. The author cites the work of Hartley with overneutralized, neutralized and underneutralized preparations. He suggests that in view of the results obtained in America and Russia with underneutralized preparations, similar German products should be employed.

SCHERER, Duluth, Minn.

EPIDEMIOLOGY OF MEASLES IN PALESTINE. S. EPSTEIN, Gsndhtsfürs. f. d. Kindesalt. **4:**411 (Nov.) 1929.

The author describes an epidemic which occurred after the war. War and military occupation had reduced the number of contacts with the surrounding countries and so allowed a generation to grow up which had not been immunized to the disease. Measles developed in about 10 per cent of the child population. The statistics are doubtlessly not complete. The higher percentage of deaths among

the females is probably due to the neglect which the girl suffers in the East. The Arab population had a higher morbidity and mortality. This is undoubtedly due to the higher social and economic position of the Jewish population. The disease assumed severer forms in the mountain districts.

REICHLE, Cleveland.

THE MEINICKE REACTION IN CONVALESCENTS FROM DIPHTHERIA. H. HENTSCHEL and L. SZEGÖ, Klin. Wchnschr. **8**:1395, 1929.

Micro-Meinicke reactions in children with diphtheria became positive by the eleventh to fifteenth day following antiserum injections in 81 per cent of the serums tested. There seems to be no relation between such positive reactions and serum sickness. Positive Meinicke reactions have been observed also with other diseases following serum injection. The serum reactions become negative gradually after several weeks. Other serologic tests for syphilis, such as the Wassermann and the Sachs-Georgi, react in much the same way following immune serum injections.

EDWIN F. HIRSCH.

THE ALLERGIC NATURE OF DICK TOXIN. W. GRUNKE, Med. Klin. **25**:1279 (Aug. 16) 1929.

Fresh serum of persons giving positive reactions to the Dick test in the amount of from 0.01 to 0.02 cc. was injected intracutaneously into persons giving negative reactions. From seventeen to twenty-four hours later, Dick toxin was injected intracutaneously in the same site. The behavior of the skin was studied during the following forty-eight hours. In order to rule out a possible inflammatory reaction, a control injection was made with the same serum. Seven positive serums and twenty-five persons giving a negative reaction to the Dick test were used in the experiments. In three negative persons the skin was made sensitive to toxin through the injection of the positive serum as described. Although the number of successful experiments was rather small, the author thinks they prove nevertheless that human skin can be sensitized to Dick toxin. Negative serums gave entirely negative results. It seems that the transmission of sensitivity is a specific reaction due to antibodies in the positive serum.

ZENTAY, St. Louis.

A NEW SYMPTOM OF GERMAN MEASLES. K. STOLTE, Monatschr. f. Kinderh. **45**:206 (Nov.) 1929.

In a number of cases of German measles, Stolte observed that the patient had a severe night sweat two days before the outbreak of the eruption. The day following this night sweat the patient was in perfect health and had absolutely no symptoms that night. The following morning the eruption appeared.

Stolte noted this condition in so many cases that he thinks it may be an important premonitory sign.

GERSTLEY, Chicago.

TYPHOID AND PARATYPHOID IN CHILDHOOD. HELMUT MOMMSEN, Monatschr. f. Kinderh. **45**:331 (Nov.) 1929.

The writer reports seventy-three cases of typhoid and paratyphoid in children under 14 years of age from 1916 to 1928.

While typhoid is usually considered as one of the diseases following Koch's laws, the author feels strongly that sufficient attention has not been paid to the studies of Pettenkofer. This writer has studied typhoid from the point of view of general epidemiology and has brought to light interesting relationships of the disease to climate and geology.

It is interesting to note that typhoid attacks the stronger rather than the weaker children and that while females show a higher morbidity, males suffer

a higher mortality. While the separation of typhoid and parathyphoid can be made bacteriologically, in childhood they run about the same course clinically. Paratyphoid is more frequent in younger children.

Juvenile typhoid shows a definite tendency to recurrence. Such recurrences are frequent but the remissions show a gradually decreasing height of the peaks of temperature.

A definite bradycardia is present in all severe cases in the beginning, which may persist for some weeks in the extreme cases. During recurrences the pulse shows a tendency to be higher than the temperature indicates, and during the convalescence, tachycardia and respiratory arrhythmia become frequent. These abnormalities in the pulse rate are probably related to disturbances of the vegetative nervous system.

As regards the gastro-intestinal tract the symptoms are characterized by extreme variations. Many children are constipated. Others have attacks of diarrhea alternating with constipation.

In sixty-nine of the children studied, sixty-one (88 per cent) gave a positive Widal reaction. Blood cultures were positive in twenty-nine (42 per cent.) In twenty-one (16 per cent), bacteria were isolated from the stool, and in four (6 per cent) from the urine.

There is no specific therapy as yet. The diet should be rational, i. e., neither underfeeding nor overfeeding.

GERSTLEY, Chicago.

THE DEVELOPMENT OF DIPHTHERIA THROUGH ANTITOXIN DEPRESSION. JOSEF SIEGL, München. med. Wchnschr. **76**:1632 (Sept. 27) 1929.

Persons who give a negative reaction to the Schick test and who have a high titer of antitoxin in their serum may develop diphtheria. This is explained by a temporary depression of the antitoxin content of the serum. Such a depression may be produced artificially by injecting small quantities of toxin intradermally. Siegl reports two interesting cases in which this was done. One was that of an infant, aged 8 months, in whom a culture from the nose, on November 19, was negative for diphtheria bacilli. Three and four days later the antitoxin titer was between 0.64 and 1.28 units per cubic centimeter of serum. On November 23 a Schick test ($\frac{1}{50}$ minimum lethal dose) was done and gave negative results on the following two days. On November 24 and 25 the antitoxin had fallen to between 0.004 and 0.02 units per cubic centimeter of serum. On November 25 a double Schick test dose ($\frac{1}{25}$ minimum lethal dose) was given intradermally. On November 26 the antitoxin titer had fallen to less than 0.004; the result of the Schick test with the double dose was positive and that of the test with the single dose became positive. A week later cultures from the nose and throat were negative for diphtheria bacilli. On the following day the antitoxin titer was less than 0.004 units per cubic centimeter of serum, the culture from the nose was positive for diphtheria bacilli and the infant developed a clinical nasal diphtheria. This responded promptly to a subcutaneous injection of 1,500 units of antitoxin. The details are readily followed in the accompanying tabulation.

	November								December			
	19	22		24		26		28	3		5	6
Culture.....	—	..	..	..	..	..	..	..	—	+	+	+
Antitoxin units per cc. of serum	..	<1.28 >0.64	<1.28 >0.64	<0.02 >0.004	<0.02 >0.004	Less than 0.004	Less than 0.004	<0.02 >0.004	..	Less than 0.004	..	..
Schick tests.....	..	..	1/50 minimum lethal dose	—	— 1/25 minimum lethal dose	+	+	..	..	Nasal diphtheria		
						+	+	+				

The other case was that of an infant, aged 4 months. Several cultures from the nose and throat were positive for diphtheria bacilli before any toxin was injected, but the infant was perfectly well. There was no reaction following either one or two Schick test doses of toxin ($\frac{1}{50}$ and $\frac{1}{25}$ minimum lethal dose) injected two days apart. However, a positive reaction was obtained with $\frac{1}{10}$ minimum lethal dose. Two days later the infant had nasal diphtheria which was controlled by the injection of 1,500 units of antitoxin. In this case a diphtheria carrier developed clinical diphtheria during an artificial depression of the antitoxin in his serum, as judged by the skin reactions to the injection of toxin. No measurements of the serum antitoxin were taken.

BRAHDY, Mt. Vernon, N. Y.

SHOULD THE DISCHARGE OF CONVALESCENTS FROM SCARLET FEVER BE BASED ON BACTERIOLOGIC EXAMINATION OR CLINICAL OBSERVATION? OTTO KLINGBERG, München. med. Wchnschr. **76**:1833 (Nov. 1) 1929.

Klingberg tried to determine the relative merits of bacteriologic and clinical criteria for discharging patients convalescing from scarlet fever. Repeated throat cultures for *Streptococcus hemolyticus* were made from 184 convalescents before they were discharged. A control group of 235 convalescents were discharged only on clinical observations. In both groups there were patients who communicated scarlet fever to others at home. The percentage of return cases was practically the same, 3.4 in those examined clinically and 3.2 in those examined bacteriologically. In addition Klingberg found that three patients who had no bacteriologic evidence of contagiousness on discharge communicated scarlet fever to contacts at home.

BRAHDY, Mt. Vernon, N. Y.

PROPHYLAXIS AGAINST INFECTIOUS DISEASES. HANS MAUTNER, Wien. klin. Wchnschr. **42**:1383 (Oct. 24) 1929.

The author states the following opinions in reference to prophylaxis:

For variola, a virus that is too strong may give rise to generalized vaccinia even in the noneczematous child. For this reason, the author advocates intradermal vaccination with 0.1 cc. of lymph diluted 1:250. This method can be safely used in the first year of life, an age at which postvaccinal encephalitis does not occur. The disadvantages are that (1) the strength of lymph must be known, and (2) no scar results as evidence of the success of such vaccination.

For measles, it is often advantageous to use one-half dose of convalescent serum, in order that the child may have mild measles and become permanently immune.

The author questions the usefulness of prophylaxis against diphtheria. He does not use toxin-antitoxin because accidents have occurred, and he feels that toxoid gives too severe reactions.

In prophylaxis against scarlet fever the results are more doubtful than in the case of diphtheria.

Active immunization against tuberculosis by any artificial method is not advocated.

CLAUSEN, Rochester, N. Y.

PROPHYLAXIS AGAINST MEASLES WITH MOBILIZED HUMAN SERUM. PAUL FERENCZ, Wien. klin. Wchnschr. **42**:1407 (Oct. 31) 1929.

The author points out that this method recently advocated by Knöpfelmacher and Baar was discussed by F. von Torday before the German Pediatric Society at Göttingen in 1923. Von Torday failed to protect 19.5 per cent of exposed children. The better results of Knöpfelmacher and Baar may have been due to the use of the method in older persons, some of whom were already immune. At any rate, the success of the method is not encouraging in view of the fact that the serum of adults who had measles as children protected all but 11.7 per cent of 125 exposed children in another of F. von Torday's series.

CLAUSEN, Rochester, N. Y.

REPORT OF THE "ALASTRIM" COMMITTEE. W. A. KUENEN, J. KUIPER and J. J. VAN LOGHEM, Nederl. Tijdschr. v. Geneesk. **2**:4937 (Oct. 19) 1929.

In October, 1929, the minister of public health instructed a committee consisting of the three authors to make a report on the epidemic known as "alastrim" which began in the middle of July, 1929, in some towns of Holland (Hannema: Cases of Alastrim in Rotterdam, abstr., AM. J. DIS. CHILD. **38**:1086 [Nov.] 1929). The first question was whether the disease was variola or alastrim. The committee first discusses the clinical symptoms of the epidemic in Rotterdam and concludes that the clinical picture of the cases observed in Rotterdam resembles that of variola, while the form of the pustules and the frequent occurrence of fever in the secondary stage are contradictory to what is accepted as typical for alastrim. The first cases which occurred in Rotterdam were not typical enough in this respect to justify the diagnosis variola.

In the second place the committee discusses the mortality. Up to October 5 296 cases occurred in Rotterdam, with 16 deaths (5.4 per cent). Of these cases 57 were in nonvaccinated patients, with 9 deaths (16 per cent). Of these 9, 8 were in children up to 10 years of age. In vaccinated patients up to 50 years of age there occurred 187 cases with not a single death. The high mortality found especially in the nonvaccinated children is in striking contradiction to what is communicated by those authors who regard alastrim as a disease quite different from variola.

It is highly probable that the disease was introduced into Rotterdam by a seaman from India.

The epidemic in Delft was quite different from that in Rotterdam: There were many cases without a single death, in the vaccinated as well as in the nonvaccinated. But the committee concludes that it is quite impossible to get a real picture of the number of cases of variola in Delft, as many cases in which only fever or pustules were present were isolated as cases of alastrim or of variola.

Further the committee is of the opinion that owing to the danger of encephalitis vaccination could not be required.

VAN CREVELD, Amsterdam, Holland.

STREPTOCOCCI IN SCARLET FEVER (SCARLET FEVER TYPE). V. RENČ, Časop. lék. česk. **66**:342 (Feb. 25) 1927.

The author presents a complete summary of the work on scarlet fever since Loeffler showed the frequency of streptococci in scarlet fever in 1895. The gram-negative quality of the scarlet streptococcoci at times changes to gram-positive when ascites agar or hemoglobin agar plates are replaced by plain agar plates. The gram-negative quality, however, remained after animal passage. A seven week isolation is advocated by some on the basis of contact cases after the six weeks' isolation. Johann reports streptococci in 97.6 per cent of cases in the first week, in 46.6 per cent in the sixth week, in 33.3 per cent in the seventh week and in 7.7 per cent of apparently healthy persons. In several series of vaccine prophylaxis cited, the best results were obtained after three inoculations, varying from 0 to 1.9 per cent. The hypotheses can be divided into the filtrable virus, streptococcus, anaphylactic and microbe (diplococcus) groups. The author advises: (1) repeated inoculation, based on the susceptibility shown by the Dick test; (2) cultures of released patients for scarlet streptococci, and (3) throat culture of contacts (family) for carriers. In this way obscure cases will be diagnosed and possibly knowledge of the etiology of nephritis and sepsis will be increased.

STULIK, Chicago.

THE ETIOLOGY OF DIPHTHERIA. S. KOPAC, Časop. lék. česk. **66**:527 (March 28) 1927.

Both essential carriers (who have had clinical diphtheria) and accidental carriers (who have not had clinical diphtheria) may be the source of virulent

diphtheria, even though culturally avirulent. From three to four negative cultures are recommended for the release of quarantine. The isolation of carriers separate from diphtheria cases is advocated. In the two cases reported, diphtheria developed soon after exposure to an obscure carrier. According to O. Seidel, forty-four hour cultures are essential for accurate diagnosis. Knebel reports from 9 to 10 per cent negative cultures after twenty hours and 100 per cent positive culture after forty-four hours in a series of 576 cases.

A bibliography is included.

STULIK, Chicago.

SCARLET FEVER AND BLOOD GROUPS. P. KISS and Z. TEVELI, Orvosi hetil. **73**:631 (June 29) 1929.

The authors studied 172 cases of scarlet fever treated at the Stefania Children's Hospital, between 1924 and 1929. They sought answers to the following questions:

1. Is there any prevalence of scarlet fever in any of the blood groups?

2. Are complications more frequent and severe in one blood group than in the others?

Children with scarlet fever seem to show a decided prevalence in group O, while groups A, B and AB remain under the average expected for these groups.

Parallel to this, complications were found in 82 per cent of children who belong to the group O, while the other three groups showed the following frequency of complications: A, 61 per cent; B, 54 per cent, and AB, 54.54 per cent.

The more severe complications occurred more frequently in group O than in the other three groups.

ZENTAY, St. Louis.

Chronic Infections

THE AVIAN, HUMAN AND BOVINE TUBERCULIN REACTIONS IN CHILDREN, WITH A REVIEW OF THE LITERATURE OF AVIAN TUBERCULOSIS IN MAN. B. M. GASUL, Arch. Pediat. **46**:67 (Feb.) 1929.

Shortly after Robert Koch isolated the tubercle bacillus, Nocard and Roux in France, and Rivolta and Mafucci in Italy showed that the organism found in fowls was of a distinct type. Morphologically it cannot be differentiated from the other types. The specific virulence test is the best means of differentiating the avian bacillus. Most authorities agree that the guinea-pig, which is very susceptible to both the human and bovine type, shows little or no susceptibility to the avian type. Rabbits or chickens, on the other hand, injected with the avian bacillus quickly succumb.

Prof. R. Lowenstein, in 1905, reported the first definite case of avian tuberculosis in man. Enlargement of the spleen is one of the most frequent observations in avian tuberculosis. The kidneys and the bone marrow are the other points of predilection. No definite tubercles are usually seen. The tubercle bacillus enters the human body through the gastro-intestinal tract. Lowenstein states that chickens with advanced tuberculosis continue to lay eggs even during the last few days of their life. He boiled eggs inoculated with the avian tubercle bacillus and then proved that all the soft boiled eggs and two out of eight hard boiled eggs still contained live tubercle bacilli. One hundred and ten tuberculous children tested at the Universitas Kinderklinic of Vienna were found to react to both human and bovine tuberculosis; none reacted to avian tuberculin. The clinical picture of avian tuberculosis in man may differ from that caused by the human and bovine type. Avian tuberculin appears to be specific only to tuberculosis caused by the avian type of tubercle bacillus.

WILSON, Norfolk, Va.

CASE OF INFANTILE GENERAL PARALYSIS. D. JUAN JUNCOSA, Med. de los Niños **30**:262 (Sept.) 1929.

A girl, aged 14 years, suffered from epileptiform convulsions, the twitching being more marked in the right arm. Her father died at the age of 45 from a cardiovascular disease which was of obscure nature but was thought not to be syphilis. The mother had three pregnancies: one child died at 7 months from meningitis, the second was a premature macerated fetus and the third is the subject of this report. The mother's blood showed a weakly positive Wassermann and a strongly positive Meinicke reaction, and her spinal fluid was negative.

The patient showed certain stigmas of somatic degeneration, such as poor dental inplantation, a high arched palate and small development. During her first five years she progressed normally except that she was smaller than the average child. At this time she developed epileptiform states in which she would remain comatose for two or three days, with tonic and clonic seizures of the right arm which persisted for five or six hours. This would be followed by partial paralysis of the right side, more pronounced in the right arm. The child gave the impression of being an imbecile; she was habitually indifferent and played with children much smaller than herself. She was unable to learn to read in the municipal school for abnormal children.

The patient had the classic Argyll Robertson pupil and the blood gave a strongly positive Wassermann and a positive Vernes reaction. The cerebrospinal fluid gave positive reaction to the Nonne-Apelt and the Pandy tests. The lymphocytes were 18 per cubic millimeter; the Wassermann reaction was strongly positive, the Vernes reaction weakly positive and the Lange test 5555544321. The patient presented a picture of advanced dementia of the globular type.

DWYER, Kansas City, Mo.

CAN AN INFANT HAVE BOTH CONGENITAL AND ACQUIRED SYPHILIS AT THE SAME TIME? ERICH HOFFMANN, Deutsche med. Wchnschr. **55**:1289 (Aug. 2) 1929.

The question has recently been brought up whether there is a definite clinical differentiation between congenital and acquired syphilis in early infancy. It has been discussed by Rietschel, who believed that an early acquired syphilis might be transferred to the infant by way of the placenta during the last moments of delivery. The author believes this to be a remote possibility. He cites the observations that syphilitic infection always occurs in the last half of intra-uterine life. The congenital infection may therefore be so recent as to permit a superinfection or a successive infection. Whichever type of infection becomes the more prominent will depend on which was the most virulent or which transferred the largest number of spirochetes. The intra-uterine transfer of organisms shortly before birth may be small, and the later cutaneous invasion may consist of a large number. Furthermore, such a superinfection may come from an entirely different source, as from a wet nurse or an attendant. Then an apparently healthy infant may develop what appears to be a primary lesion accompanied by lymphatic enlargement and this may resemble acquired syphilis in every way. It may be possible in such cases to determine a "syphilis binaria" of infants just as Tarnowsky describes for adults. In order that such a differentiation may be possible the author urges closer cooperation between the syphilologist, gynecologist and pediatrician.

SCHERER, Duluth, Minn.

MERCURIAL INUNCTIONS WITH SHAVING-BRUSH. LEOPOLD MOLL, Monatschr. f. Kinderh. **45**:293 (Nov.) 1929.

In cases of congenital syphilis in which the mother is not in contact with the infant and inunctions of mercury are ordered, Moll recommends the use of a shaving-brush. This spares the nurses the necessity of rubbing the child with their own hands, and the fact that the brush is sterilized after each treatment helps prevent pyogenic infections.

GERSTLEY, Chicago.

Internal Diseases

CONGENITAL PYLORIC OBSTRUCTION: AN EXPERIMENTAL STUDY. J. L. BRODIE, Am. J. Physiol. **89**:340, 1929.

In rats born of mothers fed on a diet with minimal antineuritic vitamin content, ten of twenty-three showed congenital pyloric obstruction. In those cases in which the obstruction was of lesser degree, or cured by atropine, polyneuritis later developed. The vagus in all cases examined showed myelin degeneration. This pyloric obstruction was much commoner in young rats of the second generation than of the first, and seven eighths of the cases occurred in males. Attention is called to the points of similarity to congenital obstruction in man.

H. E. EGGERS.

RADIUM IN TREATMENT OF LEUKEMIA. WILLIAM L. BROWN, Am. J. Roentgenol. **19**:15 (Jan.) 1928.

Frequent radium treatment for chronic leukemia is considered of value in keeping the blood picture near normal. Tonics are of value in the symptomatic treatment. Frequent blood examinations are necessary, affording an index to the indication for radium treatment and dosage. Poor results are often due to the fact that long periods between treatments produce exaggeration of symptoms and blood changes which are then little influenced by treatment. The course of acute cases is little influenced by treatment.

STULIK, Chicago.

DIVERTICULA OF THORACIC ESOPHAGUS. LESTER A. SMITH, Am. J. Roentgenol. **19**:27 (Jan.) 1928.

The author reports nine cases of diverticula of the thoracic esophagus, in only six of which were symptoms present. Two were of the lower third of the esophagus. Congenital weakness of the esophageal wall is considered an important etiologic factor. Cardiospasm was evident in only one case.

STULIK, Chicago.

THE SALTS OF MERCURY IN THE TREATMENT OF EDEMA IN CHILDREN. W. L. FUNKHOUSER, Arch. Pediat. **46**:176 (March) 1929.

Salyrgan was given to a colored boy, aged 11 years, with cardiac decompensation and general anasarca; prompt improvement resulted. A colored girl, aged 2 years, with a generalized edema and ascites and a decompensated heart was also treated with the drug, with immediate improvement. There were no toxic symptoms, and convalescence was shortened.

WILSON, Norfolk, Va.

PEDUNCULATED ANGIOENDOTHELIOMA OF THE LIVER: REPORT OF AN UNUSUAL CASE. HARRY A. SPIEGEL, Arch. Pediat. **46**:188 (March) 1929.

A white boy, aged 13 months, with a large abdomen and a severe secondary anemia was shown at laparotomy to have a large friable tumor of the under surface of the liver. The base of the tumor, at its attachment to the liver, was 7 cm. in diameter. The patient died six hours after the operation. Microscopic examination showed numerous blood spaces lined by endothelial cells, often but a single cell in thickness. Collections of endothelial cells were seen, and new vascular spaces were apparently made out. Collections of small round cells were noted. There was a familial history and there were other apparently congenital defects in the patient. The case reported was apparently congenital.

WILSON, Norfolk, Va.

INTESTINAL HEMORRHAGE AS A COMPLICATION OF PNEUMONIA. WINGATE M. JOHNSON, Arch. Pediat. **46**:193 (March) 1929.

On the fourth day of illness, the 12 year old son of a physician had a hemorrhage from the bowel amounting to at least a pint. The following day there was a tarry stool but no further bleeding. The pneumonia terminated by crisis on the fifth day and the patient made an uneventful recovery.

WILSON, Norfolk, Va.

THE SIGNIFICANCE OF ABDOMINAL PAIN IN EARLY LIFE. PAUL W. BEAVEN, Arch. Pediat. **46**:224 (April) 1929.

The most frequent cause of infantile colic is hunger. Simple overloading of the stomach rarely causes pain. Indigestion may cause colic in children of any age. Hyperirritability seen in the hypertonic child may result in abdominal pain which is relieved by atropine. Tetany in older infants and young children may give abdominal pain; calcium chloride will be effective in these cases. Constipation may cause abdominal pain, although the writer thinks abdominal pain is not commonly associated with constipation. Abdominal pain frequently accompanies parenteral infections, particularly infections of the respiratory tract.

WILSON, Norfolk, Va.

THE IMPORTANCE OF INDICANURIA, STOOL FAT AND SCHMIDT FERMENTATION TESTS IN THE PRACTICE OF PEDIATRICS. ROWLAND GODFREY FREEMAN; EDGAR GRIM MILLER, JR., and ROWLAND G. FREEMAN, JR., Arch. Pediat. **46**:269 (May) 1929.

Obermayer's method was employed to determine the amount of indican. The Schmidt fermentation test was used to estimate the fermentable carbohydrate content of the stools. The amount of fat in the stools was determined by the method of Freeman and Miller.

One hundred and sixteen children were studied for indicanuria; 51 showed indican in more than 50 per cent of the specimens examined. The fat content of the stools ranged from 3.4 to 6.9 per cent of the total stool, an average of 5.1 per cent. There were no evidences of digestion of fat. The amount of gas shown by the fermentation test ranged from 0 to 10 cc. Only one child showed gas above 4 cc. The authors believe that in the normal child indicanuria is infrequent, that the fecal fat is under 6.5 per cent and that fermentation tests produce less than 4 cc. of gas.

One hundred and thirty-nine children with symptoms were studied; 80 per cent of these showed an excess of carbohydrate, 60 per cent an excess of protein and 33 per cent an excess of fat. Dietary restrictions, if carried out conscientiously, usually result in improvement and in a reduction of the abnormalities of urine and feces.

WILSON, Norfolk, Va.

A CLINICAL INVESTIGATION OF CHRONIC COUGH IN CHILDREN. AARON CAPPER and LEON SOLIS-COHEN, Arch. Pediat. **46**:327 (May) 1929.

Thirty-one children with a more or less constant cough of six months' or more duration were studied. The majority had had a cough for one or more years. Cough is often more troublesome at night because (1) the quiet of the night makes it more outstanding, (2) the horizontal posture of sleep makes it easy for the exudate to flow to the region of the carina and there set up the cough reflex, and (3) in the wakeful state some of the exudate is expectorated or swallowed, whereas during sleep most of it drains into the respiratory system. D'Espine's sign was found in only 19 per cent, and in 40 per cent of those showing roentgenologic evidence of tracheobronchial lymphadenopathy, 42 per cent reacted positively to tuberculin. In 20 per cent sinusitis was the chief or associated cause of the chronic cough. The dry, metallic cough, which occurs

more during the day than during the night, is more likely to be due to early glandular involvement, whereas the moist cough, accompanied by expectoration and more severe at night, is usually due to an exudative condition in the lungs or to mucopus draining into the lungs when the body is in the horizontal position.

WILSON, Norfolk, Va.

CALCIUM METABOLISM IN MARBLE BONE. RANDOLPH G. FLOOD, California & West. Med. **31**:203 (Sept.) 1929.

In a case of marble bone in a girl, aged 11, the calcium metabolism was studied. The child was adaptable to wide fluctuations in blood calcium if the transition were gradual. There was reaction to sudden change.

DICKEY, San Francisco.

PNEUMONIA RECURRING TEN TIMES IN A BOY OF TWELVE YEARS. HENRY HERBERT, California & West. Med. **31**:203 (Sept.) 1929.

A case of lobar pneumonia is reported which recurred ten times in a boy up to his twelfth year. The attacks were all mild. Prophylactic treatment with pneumococcic vaccine was given and no attacks occurred during that year.

DICKEY, San Francisco.

THE COURSE, PROGNOSIS AND TREATMENT OF CHRONIC DIFFUSE NEPHRITIS IN CHILDREN. GLADYS L. BOYD, Canad. M. A. J. **21**:679 (Dec.) 1929.

The pathologic picture in these cases depends on the stage of the disease when death supervenes. The end-result, typically, is the small kidney, the capsule of which strips with difficulty and in which the microscope reveals evidence of acute and chronic inflammation in the glomerular and interstitial tissues with secondary degenerative changes in the tubules.

The cases are divided clinically into two classes: (1) those in which symptoms or signs or both follow acute nephritis; (2) those in which no suggestive symptoms of sufficient severity seem to direct attention to the kidney until chronic changes have already taken place, and in some instances not until secondary arteriosclerosis is present.

The prognosis depends on the history of recurrent attacks or the persistence of even slight symptoms. When the variation of the specific gravity in the day is less than 5 points and at night is constantly below 1.020, a severe functional incapacity is present. Values between 3 and 5 mg. per hundred cubic centimeters of creatinine indicate a degree of damage beyond the possibility of complete recovery.

The treatment is essentially preventive and consists of the early removal of foci of infection. Stress is laid on careful examination for apical abscesses in the teeth and for infections of the paranasal sinus. General symptomatic treatment is discussed.

MOORE, Omaha.

PYURIA IN CHILDHOOD. R. B. STRUTHERS, Canad. M. A. J. **21**:685 (Dec.) 1929.

The author gives a careful review of the latest views on pyelitis and particularly of the work of Dr. Chown on the pathology of pyelonephritis. Brief reports are given of fourteen cases in older children, from 1 to 11 years of age.

The author concludes that pyuria in older children is always associated with congenital or acquired malformations of the urinary tract or some infection in the neighborhood of either kidney or ureter. He believes that every child with protracted pyuria which fails to respond to the usual methods of treatment should be subjected to cystoscopic and roentgenologic examinations within a reasonable time. The extreme limit of a reasonable time he considers to be six weeks.

MOORE, Omaha.

CONGENITAL ADENOMA OF LUNG AND HYDROPS UNIVERSALIS. P. ESCH, Arch. f. Gynäk. **133**:32, 1928.

A case of congenital adenoma of the lung in an infant with universal hydrops and hydamnion is reported. The tumor is considered as a hamartoma. Five reports of similar cases were found in the literature. A common cause for both conditions could not be established.

W. C. HUEPER.

VAGINAL PAPILLARY SARCOMA IN THE CHILD. K. ADLER, Arch. f. Gynäk. **133**:100, 1928.

A papillary fibro-epithelial tumor mixed with immature muscle cells and located in the vagina of a girl, aged 2 years, is described. A review of sixty previous reports of cases and a discussion of the symptomatology and pathology of these tumors are given. They recur after removal, but form metastases only in the regional lymph nodes. The prognosis is extremely bad; only one case in which cure occurred is on record.

W. C. HUEPER.

THE DIAGNOSIS OF PYLOROSPASM. B. KEMKES, Arch. f. Kinderh. **88**:81 (Aug.) 1929.

The most important differentiation in the diagnosis is to distinguish between pyloric stenosis and pylorospasm. Pyloric stenosis exists in two forms, according to Kemkes. The first or infrapapillary form is distinguished by the presence of bile in the gastric contents and the vomitus. The second or suprapapillary form is diagnosed by means of the roentgenogram. The stomach is usually greatly dilated, and two air bubbles are seen, a large one in the stomach and a small one in the duodenum.

SANFORD, Chicago.

THE HEREDITARY PREDISPOSITION TO GOUT. J. BROCK, Arch. f. Kinderh. **88**:128 (Aug. 20) 1929.

The histories of several families who were presumed to have a tendency to gout are followed through several generations, and the literature on the subject is discussed.

SANFORD, Chicago.

HARMLESS GLYCOSURIA IN CHILDHOOD. HERBERT RAU, Deutsche med. Wchnschr. **55**:1338 (Aug. 9) 1929.

The author has had the opportunity to study a case of this uncommon condition in a child, aged 7 years. He bases his diagnosis on the fact that in this condition there is a normal regulation of the blood sugar and a persistent excretion of sugar in spite of regulation of the diet and the use of insulin. Furthermore, various clinical symptoms of true diabetes are lacking. The child was brought to the clinic because it tired easily, was undernourished and lacked appetite. Examination showed sugar in the urine; about 50 Gm. was excreted each day. There was no acetone and the kidney function was negative otherwise. The blood sugar throughout was 0.08 per cent.

By experiment it was determined that the regulation of blood sugar was normal, that the qualitative composition of the food had no influence on the amount of sugar excreted, but that this amount depended to a certain extent on the total metabolism rate. It was found that an uncle of the patient probably suffered from a similar ailment.

SCHERER, Duluth, Minn.

RELATION OF HEREDITY AND AGE TO OCCURRENCE OF JUVENILE DIABETES MELLITUS. R. PRIESEL and R. WAGNER, Klin. Wchnschr. **8**:1398, 1929.

The maximum incidence of juvenile diabetes mellitus in the thirteenth year indicates a close relationship between the number of available islets and growth.

There is always a congenital deficiency of islets. The manifestation of latent diabetes is dependent primarily on the number of available islets. Juvenile diabetes often is actually not progressive, but only appears to be so. It is hereditary like deformity. Hereditary relations were established in 27 per cent. The greater incidence (43 per cent of their cases) of heredity in diabetes among Jews is due to inbreeding. Diabetes mellitus is inherited as a recessive, as well as a dominant, character.

AUTHORS' SUMMARY [ARCH. PATH.]

THE TREATMENT OF DISEASES OF THE LIVER IN CHILDREN WITH INSULIN AND GRAPE SUGAR. ELSE KNAKE, Ztschr. f. Kinderh. **47**:502 (May) 1929.

For the first time in diseases of the liver in children, insulin and grape sugar have been used and with favorable results. In a young child a case of acute yellow atrophy of the liver which was not of the severest type cleared up, a result which is encouraging for the further use of this form of treatment in a disease which has been hopeless and in all cases of parenchymatous icterus. The theoretical basis of the treatment is as follows: The cause, pathologically adequate for the characteristic atrophy of the liver, is a collapse of the liver parenchyma as well as a functional insufficiency of the rest of the liver which occurs as the result of increased activity of autolytic ferments due to a poverty of glycogen. Only with an adequate amount of glycogen is the liver capable of carrying out in normal fashion its detoxifying, building-up and breaking-down functions.

EMERSON, Boston.

THE BLOOD PRESSURE OF SICK AND HEALTHY CHILDREN. EDDA MEYER, Ztschr. f. Kinderh. **47**:560 (May) 1929.

The blood pressure determinations in childhood have been obtained in times past by different apparatus and methods, the most important of which are Bach's Sphygmomanometer, the Riva-Rocci apparatus, Gärtner's tonometer, palpation, graphs, oscillations, auscultation and sensation. All of these things have been investigated critically by Meyer. The literature of normal blood pressure in childhood gives different values for definite age groups, weights and lengths according to the apparatus and methods used, and opinions as to physiologic variations of blood pressure vary. In studies on 320 school children from the ages of 6 to 14 years, Meyer with the help of a Riva-Rocci machine and the auscultation method determined the blood pressure curves with the following results: 1. Blood pressure increases with increasing age, height and weight. 2. A true difference in blood pressure between boys and girls was not found except that it was higher in girls from 12 to 14 years of age than in boys of the same age. 3. The diastolic blood pressure increases only a little in the course of the years, so that because of the great increase of systolic blood pressure an increase of the amplitude results. In the pathologic material of the hospital, Meyer could observe the same thing with greater variations, which are to be explained by the very different values in the unselected material. The observation of several groups of diseases has established in general no uniform condition of blood pressure. The increase of blood pressure has in most cases the value of only a facultative system, and other symptoms must be used along with it to make a diagnosis. It is not possible to draw from it a correct conclusion as to the burden on the heart nor as to several important factors in circulation, such as pulse, volume and caliber of the vessels.

EMERSON, Boston.

BRONCHIECTASIS IN CHILDREN. EELCO HUIZINGA, Nederl. Tijdschr. v. Geneesk. **2**:4606 (Oct. 5) 1929.

According to many authors, bronchiectasis is a disease which usually occurs in early youth. In their opinion this disease frequently gives a clear clinical

picture in children. The recent introduction of the roentgen diagnosis with the use of iodized poppy seed oil 40 per cent has increased the interest in the disease. The author introduces the iodized oil directly through the larynx. In order to do this the child is fixed in a recumbent position, the larynx is made visible and a catheter introduced into the trachea. The author used this method with success even in very young children without anesthesia; but one should work quickly because the iodized oil is soon coughed up. In very young children a few cubic centimeters are sufficient. The author describes several cases in which this treatment was used. In all cases bronchoscopy was also performed and this is recommended in all doubtful cases by the author. Two patients were 2 years of age.

VAN CREVELD, Amsterdam, Holland.

ROENTGENOTHERAPY OF SPASTIC COUGH AND THE EFFECT ON THE BLOOD PICTURE. K. SCHRUTZ and O. VYCHYTIL, Časop. lék. česk. **66**:604 (April 8) 1927.

Irradiating children, aged from 4 months to 6 years, the author found a decrease in the white blood cells ranging from 50 to 70 per cent, two weeks after radiation. In one case complicated with a bronchitis the white blood cells increased from 16,750 to 18,000. The decrease in one case was 86.1 per cent in lymphocytes and 34 per cent in neutrophils; and in others it was 75.8 per cent in lymphocytes, with 88.6 per cent in neutrophils; 18.5 per cent lymphocytes and 59.2 per cent neutrophils, and 62 per cent lymphocytes and 26 per cent neutrophils. In the older children there was a tendency to an absolute neutrophilia, later again replaced by a lymphocytosis. Treatment was of greatest value in those cases associated with definite adenopathy.

STULIK, Chicago.

RENAL TESTS OF CONCENTRATION AND DILUTION IN INFANTS. G. KOENIG and E. LEDERER, Orvosi hetil. **73**:1023 (Oct. 12) 1929.

Functional tests of the kidneys as to concentration or dilution of the urine can be used in infants successfully. The authors performed tests on twelve apparently healthy infants. All infants showed normal gain of weight at least ten days before the experiments. All of them were receiving milk dilutions with from 6 to 8 per cent carbohydrate. After the last feeding at 9 p. m. the urine was collected. The following morning, that is, after ten hours of thirsting, fluid was given, the amount of which was about two or three times more than the food intake on the preceding day. One-half of this amount was given through a stomach tube, the other half subcutaneously in the form of a 4 per cent dextrose solution. Urine was collected after this over a period of three and one-half hours. Weight was controlled before and after the experiment. The results of the experiment show that infants under 3 months of age have somewhat less ability to concentrate than adults, but they can dilute well. In infants over 3 months of age, the results are practically the same as in adults. The maximum of dilution is reached somewhat later in infants than in adults, and during the period of experiment only from 7 to 20 per cent of the water is excreted through the kidneys. The weight curve is not influenced by the experiments. On the whole, the methods seem to be reliable as functional tests of the kidneys.

ZENTAY, St. Louis.

Nervous Diseases

MONGOLIAN IDIOCY: THE MANNER OF ITS INHERITANCE. MADGE THURLOW MACKLIN, Am. J. M. Sc. **178**:315 (Sept.) 1929.

After surveying literature dealing with Mongolian idiocy the author concludes that there is no adequate support for the contention that the mental or physical suffering of the mother during pregnancy, the advanced age of the mother at the time of conception, reproductive exhaustion due to a large number of pregnancies

or that the father is younger or much older than the mother has any etiologic significance in the production of this disease.

Evidence is presented that these and all other environmental influences are not the cause of Mongolian idiocy but that it is due to an inherent defect and is germinal in origin.

The author claims there is no support for the statement that it is due to the presence of one pair of unit recessive factors.

The mode of inheritance appears to be much more complex. Figures are given which show a similarity between theoretical and actual results when the theoretical is based on the assumption that the disease is due to the stimulating presence in the germ cell of five pairs of recessive factors or of two dominant in four pairs of recessive factors carried in as many different chromosomes. The author does not suggest that agreement constitutes proof that this is the mode of inheritance of Mongolian idiocy.

HENSKE, Omaha.

DIABETES INSIPIDUS AND LESIONS OF THE MID-BRAIN: REPORT OF A CASE DUE TO A METASTATIC TUMOR OF THE HYPOTHALAMUS. T. B. FUTCHER, Am. J. M. Sc. **178**:837 (Dec.) 1929.

The author discusses the literature on various theories in diabetes insipidus and reports in detail a case in which diabetes insipidus was the prominent symptom and in which autopsy disclosed that this condition was due to an alveolar carcinoma of the hypothalamus, secondary to a carcinoma of the lung and without involvement of the pituitary. He states that this tends to support the view held by many that diabetes insipidus is due to disturbance of certain regulatory centers in the hypothalamus. The belief is expressed that although there may be certain centers in the hypothalamus regulating water exchange in the body, the evidence seems strong that disturbance of the action of a diuretic-antidiuretic hormone produced in the posterior lobe of the pituitary is an important factor in the etiology of the disease, possibly by sensitizing these centers or by influencing their function.

HENSKE, Omaha.

INFANTILE AMAUROTIC FAMILY IDIOCY IN TWO JAPANESE FAMILIES. F. C. CORDES and W. D. HORNER, Am. J. Ophth. **12**:558 (July) 1929.

The authors refer to the first description of amaurotic family idiocy by Tay, and to the presentation of the complete symptom-complex later by Sachs. There are two types, the infantile and the juvenile; the former begins in infancy, and the latter at the age of 6 or 7 years. In the infantile type, the first symptoms begin at 3 or 4 months of age; there is weakness of the muscles of the neck and back and the child begins to lose its vision. The eye shows the characteristic picture of the fundus. The weakness of the muscles continues, and the child rarely lives beyond 2 years of age. The essential pathologic changes are in the ganglion cells throughout the body.

The juvenile type is relatively common in non-Jewish children, while the infantile type is relatively rare. The two cases reported are of the infantile type in children of Japanese parentage.

In the first case, that of a Japanese girl, aged 3 years, the parents were first cousins. The patient was well until 10 months of age, when vision became impaired and walking became difficult. Examination showed an overweight and mentally deficient child who walked with difficulty and had trouble supporting her head. Laboratory observations were negative. Examination of the eye showed lateral nystagmus, and the fundi exhibited the characteristic picture of amaurotic family idiocy. One sister of this child (the first born) died at the age of 3 years; the cause of death was given as pneumonia. This child developed impaired vision at the age of 10 months and mental deterioration. She had difficulty in walking and using her arms before she died.

The second was that of a Japanese girl, aged 21 months, whose parents were first cousins. The first two children died suddenly at 6 months of age; the third died at 2 years of age from convulsions. The patient appeared normal at birth, but development was slow. At 18 months, she was able to sit up and feed herself. Eight days before admission, she was no longer able to sit up or to use her hands, and had difficulty with vision. Physical examination showed profuse sweating, and marked asymmetry of head. The tongue was coated and the breadth foul; the soft palate and the pillars were heavy and edematous. There were no movements of deglutition. The chest showed a marked Harrison's groove and slight beading of the costochondral junction. Both the upper and lower extremities were extremely flaccid, with little power. The fundi showed the characteristic picture. Laboratory observations were negative.

The authors comment on the consanguinity and on the predominance of females among the non-Jewish children.

W. S. REESE.

TUMORS OF THE CAUDA EQUINA. CHARLES A. ELSBERG and KATE CONSTABLE, Arch. Neurol. & Psychiat. **23**:79 (Jan.) 1930.

This study of the clinical features of lesions of the cauda equina is essentially a differential diagnosis between new growths and inflammatory lesions (neuritis) of the caudal roots. Tumors are more frequent before the fortieth year, have a more gradual onset, produce tenderness of the spines more frequently, produce a definite upper level of objective sensory disturbance in most cases, frequently have xanthochromia, coagulation, increased globulin and increased total protein of the spinal fluid, and x-ray pictures may show an enlarged vertebral canal. Neuritis occurs more frequently after the fortieth year, progresses more rapidly, frequently produces tenderness of the spines, the upper level of objective sensory disturbance is vague in 40 per cent of cases; xanthochromia, coagulation, globulin and increased total protein of the spinal fluid are rare and roentgen observations are always negative.

BEVERLY, Chicago.

STUDIES OF CEREBROSPINAL CIRCULATION BY A NEW METHOD. ERNEST SACHS, HARRY WILKINS AND CRAWFORD F. SAMS, Arch. Neurol. & Psychiat. **23**:130 (Jan.) 1930.

A review of the literature on the place, method of production and pathway of the cerebrospinal fluid is given. The evidence is overwhelming that the fluid originates both from the choroid plexus and from the perivascular spaces of the cerebrospinal axis and that it is absorbed both in the venous sinuses and in the spinal subarachnoid space. The existence of a circulation has not been definitely established. This was studied by the injection of a dye into the closed subarachnoid space of dogs and observation of its movements. The evidence indicates that there is no true circulation of cerebrospinal fluid; the dye spread by diffusion and was greatly influenced by gravity. The reduction of pressure by lumbar puncture creates an artificial circulation toward the point of puncture. If, therefore, one desires to inject serum with the idea of having it reach all parts of the central nervous system, it is more effective to use gravity and inject it into the ventricles or basal cisterna. In meningitis, for instance, repeated spinal punctures tend to spread the infection by producing artificial circulation. Drainage is more effective at the basal cisterna than in the lumbar region.

BEVERLY, Chicago.

INTERNAL HEMORRHAGIC PACHYMENINGITIS. HAROLD A. ROSENBAUM, Arch. Pediat. **46**:56 (Jan.) 1929.

A case is reported in a previously healthy girl, aged 9 weeks. At 7 weeks of age she had a "cold" which was followed in two weeks by jaundice of moderate degree and acholic stools. She then became acutely ill with fever, vomiting and

convulsive jerkings. The fontanel became tense, and lumbar puncture revealed slight increase of pressure and a xanthochromic spinal fluid which contained red cells; no bacteria were found. Through the anterior fontanel the right lateral ventricle was punctured, and about 6 cc. of apparently pure blood was withdrawn. This coagulated in eight minutes. The Wassermann and Kahn tests were negative. Bleeding time and coagulation time were normal. Generalized convulsions followed and intramuscular injections of 15 cc. of whole blood from the father were given in each buttock. Improvement began at once and the patient made a prompt recovery. The final outcome as to mental defects, etc., is problematical.

WILSON, Norfolk, Va.

IDIOPATHIC OR HYPOPARATHYROID TETANY IN CHILDREN WITH SPECIAL REFERENCE TO CERTAIN PSYCHIC MANIFESTATIONS. W. RAY SHANNON, Arch. Pediat. **46**:346 (June) 1929.

Clinical tetany may be classified into four groups: (1) infantile tetany; (2) cases due to disturbed acid-base equilibrium, comprising gastric tetany, tetany due to excessive aeration and that due to too large intake of sodium bicarbonate; (3) transitory, environmental or accidental tetany made up of occupational tetany, tetany of pregnancy and lactation, tetany with acute disease and epidemic tetany; (4) tetany due to decreased parathyroid function, comprising postoperative tetany and the idiopathic types.

In the first group, as an etiologic factor there is a practically constant low blood calcium. In the second group alkalosis seems to be the determining factor; the total blood calcium is not disturbed. The third group is comprised of an apparently totally disconnected group of cases. The fourth group has been considered by a majority of observers to be due to parathyroid insufficiency and to be either postoperative or idiopathic.

To explain those cases of tetany presenting a normal or a slightly diminished blood calcium it has been shown that the calcium exists in two forms, a diffusible and a nondiffusible form probably representing ionic and combined forms. It has been shown that the diffusible form is low even in those cases with relatively high total serum calcium; it is this form which is thought by Liu to control the nervous symptoms. It is admitted that no proof has been advanced that the lowering of the calcium level that has been observed is due to a reduction in parathyroid function. Eight cases with psychic manifestations are reported in detail. The hereditary aspect of tetany is stressed.

The author concludes: 1. Idiopathic parathyroid tetany is probably more common in children than the attention it has received would indicate. 2. It may manifest itself only at times of unusual strain. 3. Psychic phenomena may predominate in the attack and overshadow the motor symptoms, which have usually been considered the criterion for a diagnosis. 4. These psychic phenomena may manifest themselves, in other ways, such as in night terrors, toxic psychoses or total unconsciousness.

WILSON, Norfolk, Va.

PNEUMOCOCCIC MENINGITIS TREATED WITH PREGL'S SOLUTION: RECOVERY. HARRY APFEL, Arch. Pediat. **46**:516 (Aug.) 1929.

A case is reported in a girl, aged 11 years. The onset was sudden and the patient was delirious on admission the second day. The spinal fluid was cloudy. Antimeningococcic serum was given intraspinally. Pneumococci were found in the spinal fluid, and 5 cc. of the polyvalent antipneumococcic serum (1, 2 and 3) was given intraspinally; shock resulted, followed by delirium. Frequent spinal drainage was done, one month later 10 cc. of a complex hypo-iodite preparation (Pregl's solution) was given intravenously and repeated in two days later. After this the patient made a rapid recovery and was discharged as well in about three weeks. The author thinks this is the first time Pregl's solution has been employed for this purpose in New York City and suggests that it be given a trial in meningitis of pneumococcal origin.

WILSON, Norfolk, Va.

SPINA BIFIDA OCCULTA OF THE FOURTH, FIFTH AND SIXTH CERVICAL VERTEBRAE, WITH SYMPTOMS RESEMBLING POTT'S DISEASE. M. LANCE, Bull. Soc. de pediat. de Paris **27**:194 (April 16) 1929.

A girl, aged 5½ years, had six months previously begun to hold her head inclined to the left, and had complained of pain in the neck. She was awakened during sleep by the slightest movement and would cry out with pain. Physical examination revealed that her head was inclined to the left, but that it showed no rotation. Left lateral and flexion movements, but not extension, of the head were limited. The neck was short and the concavity was exaggerated. Between the occiput and the seventh cervical vertebra was a narrow depressed space in which one could not palpate the spinous processes. Pressure over the fourth, fifth and sixth cervical vertebrae caused pain. The diagnosis of spina bifida, and not Pott's disease, was made because of the absence of limitation of extension of the neck, and because of the concavity over the vertebrae where pain was elicited on pressure. Roentgenologic examination showed the presence of spina bifida of the fourth, fifth and sixth cervical vertebrae, and the posterior arcs of these vertebrae were narrowed, while their lateral arcs were wider than those of the adjacent vertebrae. Roentgen therapy was advised, but the child disappeared. After about three months she returned, much improved; there remained only slight limitation of movement when bending the head forward, and there was no pain. The author explains the temporary attack of pain as being due to a pull on the nerve roots when a marked period of growth is experienced.

BENJAMIN, Montreal.

MENINGITIS DUE TO THE COLON BACILLUS. JUAN CARLOS NAVARRO, Arch. españ. de pediat. **13**:513 (Sept.) 1929.

The pyogenic cocci are among the most frequent causative agents of purulent meningitis. Less frequently it is produced by bacilli. Infection with the tubercle bacillus is an exception.

Among the rarest causative agents are the bacilli which inhabit the intestine, among these the colon bacillus.

The author reports a case of this rare type: a meningitis due to infection with the colon bacillus, occurring in an infant aged 2 months. The onset of the disease was insidious. All symptoms of meningeal irritation were absent, and there was no hyperthermia until the final stages of the disease.

The colon bacillus was obtained in pure culture from purulent spinal fluid of three successive spinal punctures.

SCHLUTZ, Minneapolis.

THE RECENT EPIDEMIC OF INFANTILE PARALYSIS. LUIS MORQUIO, Arch. latino-am. de pediat. **23**:405 (June) 1929.

This illustrious Latin-American pediatrist and master clinician discusses the treatment of this condition which visits Uruguay in epidemic form nearly every three or four years.

Two things in the treatment are of prime importance. The early diagnosis of the condition and the early use of either convalescent serum or the serums of Flexner or of Petit, intramuscularly and intraspinally or in the cisterna.

The serums should be used in the paralytic period but are most efficacious in the preparalytic period. Many of the cases do well without much intervention.

During the acute period, repeated hot baths of 38 C. (100.4 F.), and of ten minutes' duration are of value, especially if there is much pain. Sedatives such as chloral hydrate, opiates and phenobarbital should be given during this period.

Two weeks after onset of the disease, physiotherapy should be begun, using the method of Bordier.

For the later stages of paralysis and of atrophy, hot baths, massage and electricity are indicated.

Following this, mechanotherapy and reeducation of movement, such as walking, should be used. The latter form of treatment must continue for months and years.

Finally come orthopedic and surgical procedures for the correction of deformities, such as contracture.

SCHLUTZ, Minneapolis.

TREATMENT OF CHOREA MINOR WITH INTRAVENOUS INJECTIONS OF SODIUM SALICYLATE. LOPEZ AYDILLO, Arch. de neurobiol. **9**:69 (Jan.-March) 1929.

The author has used intravenous injections of sodium salicylate with great success in the treatment for chorea minor. Ten cubic centimeters of the solution (sodium salicylate, 10 Gm.; pure dextrose, 5 Gm.; sterilized distilled water, 100 cc.) was injected every two days until complete cure obtained. The latter was effected in six cases with less than twenty injections. In two other cases, one patient showed complete recovery before the twentieth injection although the curative process was not so rapid, while the other was practically cured after thirty injections. According to the author, the choreic movements of the tongue resist the treatment more than the other symptoms. No accident was observed in any of the eight patients during the period of treatment.

NONIDEZ, New York.

CHANGES OF THE FIELD OF VISION AND THE FUNDUS IN TUMORS OF THE PITUITARY BODY. S. BIRINGER, Klin. Monatsbl. f. Augenh. **83**:153 (Aug.-Sept.) 1929.

Contrary to the opinion sometimes held, that bitemporal hemianopia and simple atrophy of the optic nerve are typical symptoms in tumors of the hypophysis, the author points out that recent literature shows great variability in fundus and field.

He gives an account of ninety-six cases examined at the Elschnig clinic at Prague. Of these, fifty-two are reported in which the correctness of the diagnosis had been established either by subsequent operation, or by roentgenogram. The cases are divided into three groups, acromegaly, dystrophy and tumors. The first two groups disclosed all the symptoms mentioned by Pierre Marie and by Froehlich. The third group offered some few symptoms of acromegaly and dystrophy besides those of the tumors. The field was normal in eight of the fourteen cases of acromegaly, temporal hemianopia with concentric narrow fields was present in two cases, coupled with amaurosis in one of them. There was one case each of homonymous hemianopia, of concentric narrowed fields of both eyes, of central scotoma and of binocular amaurosis. Six times the normal fields were accompanied by normal fundus, once by binocular simple atrophy of the disks and once by neuritis. The postmortem examination in the case of homonymous hemianopia revealed two tumors of the hypophysis, each the size of a walnut, and softening in the right hemisphere. One of the tumors had attacked the left tractus, thereby causing paresis of the left oculomotor and weakness of the right facial-hypoglossus nerves. The fundus was normal in six cases, simple atrophy was present in five cases, neuritis in one and postneuritic atrophy in two. Not considering the case of binocular amaurosis, the vision was reduced more frequently in the right than in the left eye.

Seven cases form the second group of adiposogenital dystrophy. Two of them had normal fields and fundus, one bitemporal, two temporal hemianopia with amaurosis of the second eye and one concentric narrow fields of both eyes. One case showed an atypical course of the involvement of the field. The cases of temporal and bitemporal hemianopias were coupled with simple atrophy; the one with concentric narrow fields had choked disks. Chocked disks and simple atrophy were found in a relation of 1 : 14.

The third group consists of thirty-one cases of tumors. The symptoms were chiefly those of brain tumors, with acromegaly or dystrophy present in some of them. The classification of the field disturbances is in accordance with Wilbrand-Saenger. There were four normal fields; nine had bitemporal, eight had temporal hemianopia

with amaurosis of the second eye, one had temporal hemianopia with a normal field in the second eye; and in one case of monocular temporal hemianopia, amaurosis and temporal hemianopia developed in the second eye later on. Another case began with bitemporal hemianopia and terminated in amaurosis of one eye and temporal hemianopia of the other. One had nasal hemianopia and amaurosis of the second eye; one had superior hemianopia, three had concentric narrowed field and one had amaurosis of both eyes. Of the four cases with normal fields, one showed binocular simple atrophy, one choked disk in one eye and one postneuritic atrophy. The tumors with bitemporal hemianopias showed typical disturbances in two and atypical in six cases. These consisted in extensively narrowed nasal fields of regular or irregular types, respectively, allied with considerably diminished function of the temporal fields, or of a combination of total hemianopia with hemichromatopsia of the second eye. Central scotoma was seen in one single case of hemichromatopsia; the nasal field was considerably narrowed for colors. The fundus of this group showed binocular simple atrophy in twelve, monocular in one, binocular neuritis in two, monocular in one, postneuritic atrophy in seven, choked disk in four, atrophy after choked disk and in five and normal disks in two cases.

The vision in tumors of the pituitary body varies considerably, hence the different data compiled by various authors. The compilations of de Schweinitz are compared with those of the author in a statistical table. Changes of the vision allow no conclusions regarding the development of the tumor, nor do ophthalmoscopic symptoms offer any assistance in defining its type, location or progress.

The field was normal in more than 57 per cent of the cases in the acromegaly group while temporal hemianopia was less frequent. The frequency of normal fields equals that of bitemporal and temporal hemianopias in dystrophy. Hemianopias are predominating with 32 per cent each, in the third group, that of the tumors. Bitemporal hemianopia, considered typical in tumors of the hypophysis, is not so frequent as many authors believe. It was found in twelve of fifty-two cases, or in 50 per cent if those cases are added in which temporal hemianopia was coupled with amaurosis of the second eye.

While normal disks or simple atrophy are frequent in acromegaly, simple atrophy is twice as frequent in Froehlich's dystrophy. Simple atrophy and neuritis are found in the tumor group in a relation of 13:16. Their presence, therefore, does not exclude the presence of a tumor of the hypophysis, as had been the opinion heretofore.

The visual disturbances begin in one eye mostly, in the form of a relative scotoma of the temporal side, usually in connection with the blind spot. The scotoma may become absolute and grow larger, forming later on a sector-shaped defect.

In some cases the color perception on the temporal field will gradually disappear while white perception may persist for some time. Bitemporal hemianopias are rare.

The author considers as characteristic the great undulations of color perception and vision in the temporal fields dependent particularly on the size and type of the scotoma. Temporal visual defects extend late into the nasal field, saving central vision frequently to the last. This is different in developing amaurosis when the central vision disappears prior to that of the nasal field. Involvement of the tractus or optic nerve seems to be indicated by the presence of atypical fields, or when the defect of one eye progresses in some irregular manner into the nasal field. Descending neuritis and secondary disease of the optic nerve may, of course, also lead to the appearance of choked disks, or neuritis.

K. L. Stoll.

Progressive Hypertrophic Interstitial Neuritis. Arthur Slauck, Klin. Wchnschr. **8**:927 (May 14) 1929.

The purpose of this article is to call attention to the fact that a deformed foot may not be primarily an orthopedic problem but may be primarily the result

of a neural change. It is advisable, therefore, before doing tenotomies or tendon transplants to ascertain whether the underlying trouble is not of a neurologic character. In 1912, Johannes Hoffman described the picture of progressive hypertrophic neuritis and showed that the condition consisted of a primary degeneration of the sheath of Schwann with secondary degeneration of the myelin and axis cylinders, which process tended later to ascend and involve the cord. The clinical picture is one of a chronic polyneuritis with loss of reflexes, muscle atrophy, electrical changes and sensory disturbances in the distal portion of the limb. It is noteworthy that a familial tendency of gait disturbance or foot deformity exists and that the general appearance of a foot is similar to that seen in progressive muscular atrophy or even Friedreich's ataxia. The nerves are thickened, hard and painful, and usually all peripheral nerves are involved to some degree.

Slauck reports the cases of a father and a son. The father, aged 34, said that his father and one sister were similarly affected; he himself had had trouble as long as he could remember, though the deformity never caused him much difficulty. Examination showed a high arched foot with lost achilles and patellar reflexes. The peripheral nerves were thickened, and there was diminution of electrical responses. The feet showed symmetrical atrophy, but there were no fibrillary twitchings and sensation was intact.

The son, aged 4, from early childhood presented high arched feet with rather large insensitive nerves. This insensitiveness of the peripheral nerves is so marked that the patient will complain but little when the supra-orbital nerve is stimulated with an electric current. This point alone will usually establish the diagnosis in a doubtful case.

MOERSCH, Rochester, Minn.

UNUSUAL SYMPTOMATOLOGY AND COURSE IN ENCEPHALITIS IN CHILDREN. FRITZ VON BERMUTH, Ztschr. f. d. ges. Neurol. u. Psychiat. **118**:234 (Dec.) 1928.

Bermuth reports three cases of encephalitis in children, the first two in a brother and sister. The first case was in a boy, aged 2¾ years, who was taken suddenly ill with vomiting and an inability to walk or stand. In addition, he had a left abducens paresis, nystagmus, especially on looking to the left, conjugate gaze to the right, increased tendon reflexes and a positive Babinski sign. A left cerebellar localization was made and tumor suspected, but in view of the rapid onset, the rapid disappearance of all signs in a relatively few days and the hypersomnia, a diagnosis of encephalitis was made involving the pons and the floor of the fourth ventricle, a diagnosis which seemed to be supported by case 2, the patient in which was a sister of the first patient. This patient was a girl, aged 6, who was suddenly taken ill with vomiting, frontal headache and complete amaurosis without fever. The pupillary reactions were normal; the patella and achilles reflexes were difficult to obtain, and the eyegrounds showed a marked choking of the disks. Lumbar puncture gave normal results, with a pressure of 190 mm. of water, but cistern puncture showed a markedly increased pressure. Because of this and the complete blindness which had to be attacked in some way, the ventricles were emptied. A few days later, the child could count fingers, and in the course of a week vision had returned to 5/20 and 5/15. Though tumor was suspected, a diagnosis of encephalitis was made, with edema of the brain and a blocking of cerebrospinal fluid. The third case concerned a child, aged 8, who had an encephalitis following an attack of measles. After eight weeks, she recovered, but developed a character change characterized by lying and stealing. The mentality was decreased. During the course of a year the behavior disturbances disappeared, the mental deficit cleared up and the child became again normal. This case of metencephalitis was considered exceptional in its course.

ALPERS, Philadelphia.

BARKMAN'S REFLEX IN CHILDREN. S. TSUJI, J. Pediat. (Tokyo), no. 352, Sept. 20, 1929, p. 1612.

The author examined 103 children between the ages of 2 months and 15 years, and found the presence of this reflex in all cases. Barkman's reflex, as first described by him in 1923, consists in stimulation of the skin on one side between the nipple and the seventh interspace, which is followed by a contraction of the rectus muscle on the same side. This was found to be absent in five children — the first with a history of a fall from a bicycle, the second with typical clinical symptoms of encephalitis, the third with a history of cerebral concussion following a bicycle accident, the fourth with a history of injury to the hip and the fifth with tonic twitching of both extremities.

KATO, Los Angeles.

ACROCONTRACTURE OF PARKINSON. JAN BABICEK, Časop. lék. česk. **65**:1156 (July 30) 1926.

The author describes his observations in a boy, aged 14, who had had encephalitis about three and a half years before. He made a slow recovery, but his school work progressively declined and he developed a constant thirst. The intention tremor was progressive, and spasticity gradually became more intense. Under treatment with scopolamine the general spasticity disappeared, and the boy was able to walk alone. Babinski's sign was negative, but there was an Achilles clonus on the left side. It is unusual to find this syndrome following encephalitis in so young a patient. It is also uncommon to have the spasticity overshadow the tremor, and to have an Achilles clonus. The condition developed as a primary spasticity and shortening of the muscles with a rapidly progressing deformity, without pain or muscle atrophy. Roentgen observations were negative.

STULIK, Chicago.

ATTACKS OF FALLING WITH EFFECTIVE INCREASE OF MUSCLE TONE. K. KELLER, Orvosi hetil. **73**:625 (June 29) 1929.

The author reports a peculiar form of falling attacks that do not seem to fit in either with epilepsy or with narcolepsy. A little girl, aged 12, showed retarded mental development. She began to walk at 3½ years and the attacks of falling started at the age of 4. The attacks were described as "lightning-like;" the child dropped to the ground every time there was any affective change. The attacks were accompanied with a general increase of tonus of all muscles of the extremities and the trunk. She did not lose consciousness, and the whole attack lasted only a few seconds. The author thinks that the attacks developed on the basis of a striopallida lesion and that they can be differentiated from narcolepsy because of the presence of an increase in tonus of the muscles.

ZENTAY, St. Louis.

Diseases of the Ear, Nose and Throat

THE PREVENTION AND TREATMENT OF COMMON COLDS IN CHILDREN. WARREN B. DAVIS, Arch. Pediat. **46**:172 (March) 1929.

Local prophylaxis consists in clearing up the infections (of nose or throat) and removing or decreasing nasal obstructions.

The general plan of treatment consists in:

1. The early administration of a laxative.
2. A hot mustard foot-bath, with rest in bed and isolation.
3. A light diet with an abundance of water.
4. Atropine sulphate in small doses at from three to six hour intervals.
5. The internal administration of sodium bicarbonate or other alkalinizing agent.
6. Liquid petrolatum and a 10 per cent solution of mild silver protein instilled into the nose.

7. Instead of plain petrolatum, a solution consisting of 1 grain (0.06 Gm.) of menthol or camphor and 1 ounce (28.3 Gm.) of liquid petrolatum may be used in the nose.

8. The reactionary congestion following the use of epinephrine or ephedrine makes their employment inadvisable. Sometimes cocaine, 1 grain to the ounce of liquid petrolatum, may be used locally every six hours. Camphor ice about the anterior nares and warm salt solution or an alkaline gargle may be employed several times daily. From 2 to 5 per cent of a solution of mercurochrome-220 soluble applied to the posterior and lateral walls may be beneficial.

Cooperation between the various physicians and the public health officials is suggested.

WILSON, Norfolk, Va.

UNUSUAL FOREIGN BODY IN THE LEFT MAIN BRONCHUS OF A CHILD FIVE YEARS OLD. JACOB SOBEL, Arch. Pediat. **46**:217 (April) 1929.

A boy, aged 5 years, was found to have a foreign body (otoscope bulb) in the left bronchus at the level of the sixth dorsal vertebra. The history was not suggestive of a foreign body but rather of trauma due to pummeling administered by three boys who had sat on his chest the day previously. Abdominal pain was a prominent symptom, although the classic symptoms of foreign body were present. The picture was obscured by the presence of cellular emphysema and a partial localized emphysema due to laceration of the bronchus, pulmonary tissue and the parietal pleura with the passage of air into the surrounding tissues.

WILSON, Norfolk, Va.

LUNG ABSCESS FOLLOWING TONSILLECTOMY: ITS ETIOLOGY AND PREVENTION. HARRY APFEL, Arch. Pediat. **46**:231 (April) 1929.

The writer reviews the history of abscess of the lung and gives the bacteriology, the symptomatology, differential diagnosis and treatment. Emphasis is placed on the thoughtful preoperative and postoperative management of the patient who undergoes a tonsillectomy. The patient should be warned not to use the muscles of the throat for needless talking.

WILSON, Norfolk, Va.

MASKED OTITIS MEDIA AND MASTOIDITIS IN INFANCY. EDWARD S. BABCOCK and J. ROY JONES, California & West. Med. **31**:90 (Aug.) 1929.

The symptoms in this condition are referable to some distant part of the body rather than to the ear. Enteritis is usually present, cough, anorexia, snuffles, vomiting, fretfulness, convulsions, extreme toxicity and occasionally paralysis are among the symptoms. There may be no fever or leukocytosis. The ear-drum is more often dull gray or creamy white, with little or no bulging, than it is red and bulging. With walled-off pus in the mastoid antrum there is usually a sag of the posterior superior canal wall adjacent to the drum. These drums are best seen with an electric otoscope and small speculum. Repeated myringotomies may be necessary to maintain drainage of the middle ear. Mastoid antrotomy is indicated (1) when repeated myringotomies for purulent otitis fail to cause improvement, (2) when the infant is markedly toxic in spite of free drainage of the middle ear, and (3) in the toxic infant when examination of the ear suggests infection although myringotomy reveals no pus in the middle ear. The feeding and symptomatic treatment will vary with the type of case. The authors report several selected cases to illustrate the symptomatology and treatment.

DICKEY, San Francisco.

DEAFNESS AND THE CHILD. LAWRENCE K. GUNDRUM, California & West. Med. **31**:134 (Aug.) 1929.

Measurements were taken of a series of patients with impaired hearing before and after the removal of tonsils and adenoids. The audio-amplifier tests were made

immediately before the tonsil and adenoid operations. Some patients were reexamined one month after operation, whereas others could not be located or reexamined until eighteen months later; 8.7 per cent were definitely worse, showing less hearing function than before operation; there was no change in 3.7 per cent, and 87.5 per cent showed improvement. The greatest increase was generally in inverse ratio to the age of the patient; the younger the patient, the greater the benefit. Those with markedly enlarged tonsils and adenoids were usually much more improved. In general, the improvement in the hearing of children after the removal of tonsils and adenoids was more marked than was expected.

DICKEY, San Francisco.

THE CARE OF ACUTE OTITIS MEDIA. IRA E. GASTON, Northwest Med. **28**:419 (Sept.) 1929.

This is a review of the subject of otitis media, its complications and treatment. The prevention of otitis media is the prevention of common colds, exanthems, influenza and sinus disorders. Swimming, vigorous blowing of the nose, improper douching of the nose and intranasal operations are also exciting causes. Predisposing causes are adenoid growths, lack of tubal patency and a hereditary catarrhal tendency. Anything favoring adequate nasal ventilation, especially during colds, is of value in preventing otitis. Attention is called to the etiologic relation of mastoiditis to the choleriform intoxication of infants.

SCOTT, Philadelphia.

SURGICAL MANAGEMENT OF BILATERAL CONGENITAL OCCLUSION OF THE POSTERIOR NARES. V. K. HART and C. N. PEELER, Southern M. J. **22**:1087 (Dec.) 1929.

The authors describe an ingenious operative procedure for the restoration of permanent openings of the posterior nares.

The infants with bilateral complete occlusion inevitably develop sinus infection, the antrums and the ethmoids being present at birth. Therefore operation should be done as soon as is compatible with safety.

SCHLUTZ, Minneapolis.

CONGENITAL AUDITORY IMPERFECTION (CONGENITAL WORD-DEAFNESS); WITH REPORT OF A CASE. C. WORSTER-DROUGHT and I. M. ALLEN, J. Neurol. & Psychopath. **9**:193 (Jan.) 1929.

Congenital word-deafness is a rarity, and its inevitable disorganization of speech mechanisms has often led to misinterpretation of the underlying defect. A case is presented in detail in which the outstanding defect is the inability to appreciate the significance of spoken words; but, in addition, there is also considerable inability to distinguish between less specialized sounds. The authors therefore suggest the new term, "congenital auditory imperfection," to cover this condition for which "word-deafness" is too limited. It was evident early in this case that there were no defects in the peripheral organs of hearing and sight, and still less in the muscles of articulation and expression. At the age of 5, the patient went to a deaf and dumb school and learned some lip reading. Only after this did he use any words spontaneously. The authors recognize the futility of trying to place the different parts of the speech mechanism in separate and clearly defined "centers"; they accept the complete interdependence of each speech element, but consider it desirable, for simplicity in discussion, to use the old term "center" without implying the existence of a definite anatomic unit. In the absence of a "word memory center" it is evident that, with the perfection of lip reading, the boy will have to build up, from his memories of lip movements and facial expressions and their association with persons, objects and affective states, a new "center" for the memory of words which may be called the "lip reading word-memory center." This has been partially accomplished. There has been developed in this case a method of expression which is of the nature of so-called "idioglossia"—

an individual language representing an extreme form of mispronounced and ill expressed conventional language. As regards causation, the balance of evidence is in favor of a biologic variation or of local aplasia, but birth injury cannot be definitely excluded. Important questions grow out of this type of case such as the influence of this defect on general intelligence, the resulting abnormal psychology, the relation to delinquency and the importance of special educational methods to bring such patients to a self-supporting status.

FAVILL, Chicago.

CONGENITAL AUDITORY IMPERCEPTIONS (CONGENITAL WORD-DEAFNESS): INVESTIGATION OF A CASE BY HEAD'S METHOD. C. WORSTER-DROUGHT and I. M. ALLEN, J. Neurol. & Psychopath. **9**:289 (April) 1929.

The investigation of a case of congenital auditory imperception by Head's method is described in detail. In a previous communication describing the case, word-meaning deafness was shown to be the most important element of the defect, but some degree of word blindness and agraphia was demonstrated as well as a pronounced speech defect of the nature of so-called idioglossia. In order to distinguish between the patient's actual word hearing and his ability to lip read, the method was varied in one particular from that employed by Head for the investigation of cases of aphasia. Each test is shown in tabular form and the capacity and defects of the patient both on the "receptive" and "executive" sides are summarized. The deficient background of congenital aphasia is contrasted with that of acquired aphasia, and an estimate is made of what the patient has been able to acquire in the form of language. This application of Head's method disclosed rather more than the ordinary routine method and emphasized more definitely the salient features of the disorder. It is doubtful if Head's method will prove of value in congenital aphasia, as, in the majority of such cases, means of communication with other individuals is so imperfect and the background of knowledge so deficient that any method of approach has of necessity to be very simple.

FAVILL, Chicago.

TONSILLOMYCOSES. ALDO CASTELLANI, Practitioner **124**:67 (Jan.) 1930.

The term tonsillomycosis is used to denote any affection of the tonsils due to fungi. The author refers to a fairly complete botanical description of fungi found in tonsillomycosis from his lecture on "Fungi and Fungous Diseases," Chicago, 1926. He points out that mycetes or fungi differ from bacteria principally in that they are of larger dimensions and always reproduce by spores. The pathogenic fungi in man may be separated into two large groups: the budding fungi (yeasts and yeastlike organisms), and the filamentous fungi.

From a clinical point of view, tonsillomycoses may be separated into two large groups: (1) the acute tonsillomycosis, and (2) the subacute and the chronic tonsillomycoses.

The acute group contains two principal types: (1) the follicular and (2) the membranous. The follicular type (tonsillomycosis follicularis) in the majority of cases is due to the yeastlike fungus of the genus *Monilia* (tonsillomoniliasis). There are several whitish-gray or whitish-yellow spots on the surface of the tonsil, corresponding to the openings of the follicles. The patient complains of sore throat and discomfort on swallowing, and there may be fever. The prognosis is usually excellent.

Treatment consists of local application of glycerin of borax (B. P.), or of solution of phenol 1:20; in some cases a diluted tincture of iodine (1:2 or 1:4) is of distinct help. In persistent cases, potassium iodide should be given.

The membranous or diphtheria-like type (tonsillomycosis membranicia) is usually caused by yeastlike fungi of the genus *Monilia*, mainly of the *Monilia metalonidnensis* type. The onset is often sudden, with severe sore throat and difficulty in swallowing. The patient feels quite ill and may have rheumatoid pains in the joints. The temperature may reach from 102 to 103 F. There are

cream-white patches on the tonsils, uvula and occasionally the soft palate. The patches lack the elasticity and resiliency of a diphtheria pseudo-membrane.

The prognosis is generally favorable, but not always. The condition may spread to the bronchi and lungs, and a severe bronchopneumonia may develop.

Tincture of iodine should be applied to the patches, and a carbolic spray (1 per cent) may be used. Potassium iodide given internally may be an aid.

Diagnosis of both the acute types is based on the microscopic and cultural examination.

The principal types of subacute and chronic tonsillomycoses are: (a) tonsillo-actinomycosis, (b) tonsillopseudo-actinomycosis, (c) tonsillomycosis fusa and (d) tonsillomycosis spiculata vel spinulosa.

Tonsillo-actinomycosis is quite rare and is caused by fungi of the genus *Nocardia (Actinomyces, Discomyces, Streptothrix)*. An abscess forms slowly in the tonsil and peritonsillar tissue. The abscess opens spontaneously and discharges pus containing yellow or whitish-yellow grains. The abscess may continue discharging indefinitely. Progress is good if the patient is treated early. Potassium iodide in large doses (30 grains [1.9 cc.] to 4 cc., three times daily) is efficacious.

The pseudo type is similar to tonsillo-actinomycosis, but the pus does not contain grains, and it is less influenced by potassium iodide. The tonsillomycosis fusa is a mycotic condition of the tonsils and surrounding tissues. It usually runs a chronic course. There are peculiar brownish patches on the surface of the tonsil. The diagnosis is made by microscopic and cultural examinations. The prognosis is good as to life, but the patches may persist for months or years. Administration of diluted tincture of iodine locally and of potassium iodide internally may be of value.

Tonsillomycosis spinulosa runs a chronic course. On the surface of the tonsils one may see numerous gray, grayish-brown or white erect spicules, occasionally formed in bundles originating in the crypts. The diagnosis is based on microscopic and cultural evidence. The treatment is the same as for the other chronic forms.

SAPPINGTON, San Francisco.

THE SURGICAL ANATOMY OF THE TONSIL AND ITS BEARING ON COMPLETE TONSILLECTOMY. R. SCOTT STEVENSON, Practitioner **124**:82 (Jan.) 1930.

A list of diseases which has been attributed to infection of the faucial tonsils reads like the index of a textbook in medicine. Nevertheless, there are a large number of conditions in which tonsillectomy has been approved by competent authorities. All agree that if tonsillectomy is indicated, the tonsils should be removed completely.

The faucial tonsil is not a lymph gland covered with a capsule as it has frequently been described, but it consists of a mass of lymphoid tissue embedded in a triangular fossa formed between the glossopalatine (anterior) and pharyngopalatine (posterior) pillars of the fauces and the side of the tongue. The tonsils are developed from the dorsal angles of the second pharyngeal pouches, and the endoderm lining these pouches grows in the form of solid buds into the surrounding mesoderm. These buds are hollowed out by the degeneration of their central cells and form the crypts of the tonsils. Lymphoid cells accumulate around the crypts and group themselves to form lymphoid follicles. The faucial tonsil is therefore a somewhat tubular structure, with a hoodlike upper lobe surrounding the so-called supratonsillar fossa, a central lymphoid mass and a lower lobe. A fold of mucous membrane arches backward from the glossopalatine pillar, and the upper part of this fold, the plica semilunaris, stretches between the two pillars. The lower part of the fold, the plica triangularis, forms the medial and lower parts of the tonsil, which it overlaps. Denis Browne has described a "suspensory ligament" connecting the anterior angle of the tonsil to the base of the tongue. This band of fibrous tissue is sometimes of importance in the surgical removal of the tonsil. The deep surface of the tonsil is separated from its muscular bed by some loose areolar tissue, which is easily split into layers.

The prominence of the anterior pillar of the fauces is formed by the palatoglossus muscle which has a fanlike origin from the anterior surface of the soft palate. The superior constrictor muscle forms the circular musculature of the pharynx. In its inner surface it is related to the palatopharyngeus muscle and the pharyngeal aponeurosis; it is usually described as being also related to the capsule of the tonsil, but careful dissection shows that the palatopharyngeus separates the tonsil from the former muscle.

Part of the palatopharyngeus muscle forms the posterior pillar of the fauces and defines the bed of the tonsil behind. Certain fibers of it are attached to the deep surface of the tonsil which makes it almost impossible to remove the tonsil without removing a certain portion of the muscle fiber also.

In dissecting out a tonsil the surgeon finds it quite easy to strip the upper pole from its bed until about two-thirds the way down the tonsil he encounters the muscular fibers which enter the tonsil from the palatopharyngeus. The usual procedure is then to arrange a snare around the tonsil and to trust to the action of the snare to remove the lower pole of the tonsil. If the muscular fibers are few and unimportant, this procedure is quite successful, and the tonsil is completely removed; however, if the muscular fibers are more abundant, the use of a snare to complete the enucleation must inevitably leave a large piece of lymphoid tissue behind. The same results are usually obtained by using a guillotine instead of the snare.

The author believes that to remove the tonsil completely in 100 per cent of cases, a careful, methodical, slow blunt dissection should be employed. He states that by this method not only is it certain that every tonsil can be completely removed, but in most cases the operation can be made practically bloodless, the pillars of the fauces are not damaged, the pain after the operation is greatly lessened, and the period of convalescence is shortened. The disadvantages are that it is not easy, it takes longer, and it requires a longer and deeper anesthesia.

SAPPINGTON, San Francisco.

ADENOIDITIS AS A CAUSE OF PROLONGED FEVER IN CHILDREN. T. VALLEDOR and A. CODINACH, Bol. d. Soc. de cubana pediat. **1**:621 (Nov.) 1929.

Six cases are reported in patients whose age varied from 6 to 14 months, and in whom the temperature was between 37.2 and 37.5 C. (98.9 and 99.5 F.) for periods of from twenty days to seven months. The only symptoms in these cases were the temperature and some slight nasal discharge. Operation and treatment for adenoids gave relief from both these symptoms. The treatment consisted of nasal drops or sprays of sodium chloride, epinephrine, ephedrine inhalent, or occasionally 5 per cent of a brand of mild silver protein. Operation is simple if done by a qualified person. It can be done after one year.

SANFORD, Chicago.

ACUTE MASTOIDITIS: PROBLEM OF "EARLY" OPERATION. P. HOPSTEIN, Monatschr. f. Ohrenh. **63**:30 (Jan.) 1929.

Of 686 patients with acute otitis media who came under his observation at the St. Maria-Viktoria Clinic in Berlin, Hopstein reports that 383 were healed by conservative treatment. Most of the patients had been under treatment before entering the hospital. The 303 patients operated on were in many cases in a serious condition necessitating immediate intervention. In fourteen cases the time of onset of the symptoms could not be traced, these were excluded from the statistics. In 82 of the remaining 289 patients, more or less severe complications were present. Sixteen patients died; however, in one case death was due to an intercurrent infection with scarlet fever and in another to tuberculous meningitis. The mortality for otogenous disease in acute mastoiditis was therefore 18 per cent, as compared to 28 per cent in cases with complications. For the purpose of discussing the question of "early" or "late" operation, Hopstein grouped his material in the following manner: In the first group of twenty-three patients,

operation was performed during the first week of the disease. In six instances perforation had occurred. The mortality was 4.3 per cent as compared to 80 per cent in complicated cases. The following group of eighty-seven patients contained founteen patients with perforation. In all these antrotomy was done during the second or third week of the disease. The mortality was 5.7 per cent, as compared to 18 per cent of patients with complications. Operation after the third week of the disease was performed on 179 patients; in 61 of these, perforation had occurred; 59 had complications. The mortality was 4.5 per cent as compared to 33 per cent in cases with complications. On the basis of his experience Hopstein considers the term "early" operation should be set aside. Operation in clinically mild cases solely on the basis of pain in the mastoid process and an increased temperature with otherwise normal condition is not justified. However, if the condition of the ear or general state of the patient warrants intervention one should not hesitate to intervene by antrotomy.

[ARCH. OTOLARYNG.]

PALATINE TONSILS: DEPOSITION OF PIGMENT CONTAINING IRON AND PIGMENT WITHOUT IRON. H. BRUNNER, Monatschr. f. Ohrenh. **63**:141 (Feb.) 1929.

On the basis of a review of the literature and his personal study on twenty-five pains of tonsils derived from tonsillectomies and fourteen pains from necropsies, besides experiments on eleven rabbits, Brunner states that under normal conditions the tonsils have a low iron content. Iron is more often evident in material obtained from necropsies than in tonsils derived from tonsillectomy. It is accumulated on the borderline between lymphatic tissue and the capsule, in the lymphatic tissue itself and is also toward the outside of the follicle. Pigment without iron is found on the whole, in the same places, as pigment containing iron. Deposition of iron and pigment is most frequently due to resorption of a pathologic hemorrhage caused by trauma or an inflammation. It may also be associated with hemolysis occurring in the tonsils under certain conditions. Iron derived from the blood circulation is likewise deposited. Following intravenous injection of a small amount of colloidal iron into rabbits, the iron is first deposited in the wall of tonsillar veins, then on the wall of the capillaries. In this respect they are to be distinguished from the lymph glands. However, as the endothelium of vessels in the tonsils is impermeable for colloidal iron and accumulation of iron in the reticular cells is thus prevented, the tonsils resemble lymph glands, but are at variance with the behavior of the liver, spleen and bone marrow.

[ARCH. OTOLARYNG.]

OTITIS AS COMPLICATION OF PNEUMONIA IN CHILDREN. L. HOFMANN, Monatschr. f. Ohrenh. **63**:237 (March) 1929.

On the basis of his experience, Hofmann states that acute otitis media is a frequent symptom accompanying pneumonia in children. The proportion was 3 to 1. Distribution according to age was irregular; however, he found most cases in children under 4 years of age. There was no fundamental difference in the course of otitis in lobar pneumonia or bronchopneumonia, although it was seen more frequently as a complication in the latter disease. In 179 cases, 58 children developed otitis media, whereas in 275 cases of lobar pneumonia disorders of the ear were evident in only 44. Otitis media acuta simplex (or acute otitis media catarrhalis) was observed in 52 cases (or 19 per cent of all cases of otitis). Purulent acute otitis media was observed in 243 cases (or 81 per cent). Its occurrence was seen only in 12 cases as a precursor of pneumonia, and it usually ceased rapidly. Likewise rarely was this disease seen on outbreak of pneumonia; only 18 cases were reported; however, it frequently persisted for a long time. As a complication of pneumonia, 191 cases demonstrated its frequent incidence. Its course was the most favorable. Indication for surgical intervention is rarely found. Otitis in this case may be classed among the symptoms of "second sickness" occurring after pneumonia, such as sore throat, pharyngitis, lymphadenitis and bronchitis, and expectant treatment is indicated.

[ARCH. OTOLARYNG.]

Diseases of the Skin

CONGENITAL ECTODERMAL DEFECT. A. W. FALCONER, Lancet **2**:656 (Sept. 28) 1929.

The author describes a boy, aged 6½ years, born in Zululand, whose appearance simulated closely that of a diminutive old man. The boy had only two teeth. The head was covered with almost colorless down and there was complete absence of eyebrows. The whole skin was dry but not harsh. The lower jaw was poorly developed. The Wassermann reaction of the blood was negative; nevertheless in view of the tabes found in the father, congenital syphilis was considered. In many respects the case resembled the clinical picture described by Hastings Gilford as progeria. Harvey Cushing definitely accepts progeria as a pituitary syndrome.

LANGMANN, New York.

THE DIETARY REGIMEN IN THE TREATMENT FOR INFANTILE ECZEMA. L. RIBADEAU-DUMAS, R. MATCHIEU and W. CLOG, Paris méd. **2**:491 (Dec. 7) 1929.

The underlying metabolic disturbance seems to be a change in ratio between the serum proteins, particularly the albumin and globulin fraction. There is also trouble in water metabolism. For these reasons, proteins have been under suspicion, and animal protein in particular. The authors believe that the milk fat is the cause of more trouble, than the proteins. Various forms of nonmilk or nonanimal protein diets are discussed, but the authors do not think that they are of much value, their contention being that the child has need of milk proteins for growth, and that the elimination or reduction of these proteins is of doubtful value. Fresh air, particularly sea air, and altitude help wonderfully, and the condition is inclined to be self-limiting.

SANFORD, Chicago.

Surgery and Orthopedics

APPENDICITIS IN CHILDREN. CHARLES E. FARR, Arch. Pediat. **46**:335 (June) 1929.

The diagnosis is discussed in detail. In 805 cases at St. Mary's Free Hospital for Children the total mortality was 51, or 6 per cent. There was no mortality in the chronic cases. For the acute cases the mortality was nearer 8 per cent. The only case occurring in infancy resulted in death.

In 654 cases at the New York Hospital in the First Surgical (Cornell) Division from 1915 to 1928 inclusive, the total mortality was 3.7 per cent; 154 cases were chronic with no deaths; 500 were acute with a mortality of 4.8 per cent and 243 acute cases were closed without drainage (early cases) with no deaths. In 302 consecutive acute unperforated cases, there was no death. The total mortality in the two services, numbering 1,459 cases, was 75 or 5.2 per cent. As there was no mortality in the chronic cases the percentage in the acute cases was nearly 6 per cent. This is higher than a similar series in adults would yield. At the New York Hospital, where comparatively few young children were treated, the mortality in the acute cases was 4.8 per cent. In children over 5 years of age the mortality in 579 consecutive acute cases was 3.2 per cent; this was superior to their results in adults. In children under 5 years of age the mortality was 13 per cent. The principle factor in the high mortality in children under 5 years was delay in operation due to difficulty in accurate diagnosis. In this series few children under 5 years of age with chronic appendicitis were operated on, probably because the symptoms were usually overlooked until a definite attack occurred.

The following summary is given: 1. Appendicitis is common in childhood. 2. The underlying anatomic changes in chronic or recurring appendicitis are usually present from birth. 3. The difficulty of diagnosis is great but not insurmountable. 4. Chronic appendicitis causes practically no operative mortality in childhood. 5. Early diagnosis in acute cases and early operation yield just as good results in children as in adults.

WILSON, Norfolk, Va.

AN INTERESTING CASE OF MECKEL'S DIVERTICULUM ASSOCIATED WITH INTESTINAL HEMORRHAGE, DIVERTICULITIS AND ACUTE INTESTINAL OBSTRUCTION WITH RECOVERY. JOE T. SMITH and OLIVER W. HILL, Arch. Pediat. **46**:521 (Aug.) 1929.

Twenty-six cases with intestinal hemorrhage have been reported. A boy, aged 14 months, who had previously been well, had two rather large stools with a considerable amount of dark red blood. The night of the fourth day he went into shock and when seen was toxic and uncomfortable. The results of a physical examination were essentially negative except for slight abdominal distention and palpable peristalsis over the abdomen. Operation revealed a gangrenous perforated Meckel's diverticulum, about 4 cm. in length, well above the cecum. The patient made a slow but gradual recovery. All cases of obscure intestinal hemorrhage should be watched closely for indications of this condition. Twenty-six of the twenty-seven cases reported occurred in boys.

WILSON, Norfolk, Va.

ACUTE TORSION OF AN UNDESCENDED TESTIS IN AN INFANT AGED ELEVEN MONTHS. EDWIN A. RIESENFELD, Arch. Pediat. **46**:524 (Aug.) 1929.

A case is reported in a child aged 11 months. Examination disclosed a hot, red, tender mass, about the size of a walnut, in the left inguinal region. The gangrenous testicle and epididymis were removed. After the operation an uneventful recovery occurred.

WILSON, Norfolk, Va.

ANESTHESIA FOR CHILDREN, WITH REFERENCE TO ORTHOPEDIC SURGERY. JAMES RAYMOND MARTIN, California & West. Med. **32**:93 (Feb.) 1930.

The case records of 1,807 children given anesthesia over a period of four years at the Los Angeles Orthopedic Hospital were examined. The series began with a high percentage of ether anesthetics; later, anesthesia with nitrous oxide was in the lead. During the last eighteen months of the time, ethylene for anesthesia was used almost routinely. It was concluded that none of the anesthetics should be used routinely in orthopedic cases, but the one of most merit and best suited for the individual case should be used. The physical and mental condition of the child on entering the operating room, the care and skill of administering the anesthetic, and the length of time and severity of the operation are all important points to be noted. The proper handling of the patient, both before and during the anesthetic, is as important as the type of anesthetic used. As most orthopedic operations are not emergency operations, the operative work should be done at the time when the patient is in the best possible physical condition.

DICKEY, San Francisco.

INCARCERATED DIAPHRAGMATIC HERNIA IN AN INFANT, WITH OPERATION AND RECOVERY. RALPH B. BETTMAN and JULIUS H. HESS, J. A. M. A. **92**:2014 (June 15) 1929.

An infant, aged 3½ months, with an incarcerated diaphragmatic hernia was operated on; the hernia was reduced and the diaphragm repaired. Both abdominal and intercostal incisions were necessary. The ribs forming the arc of the diaphragmatic defect were fractured, allowing an imbrication of the diaphragm. Intrathoracic drainage was not used. This appears to be the earliest successful operation for diaphragmatic hernia.

BONAR, Salt Lake City.

MECKEL'S DIVERTICULUM ASSOCIATED WITH SEVERE HEMORRHAGE. EDWIN G. SCHWARZ and JACK DALY, J. A. M. A. **93**:1468 (Nov. 9) 1929.

This is a report of another case of Meckel's diverticulum in a boy aged 8, in which hemorrhage from the bowel was encountered. The increasing frequency of

these reports emphasizes the fact that hemorrhage from the bowel is a not uncommon symptom of ulcerative Meckel's diverticulum.

BONAR, Salt Lake City.

HERNIA OF THE DIAPHRAGM IN CHILDREN. P. E. TRUESDALE, J. A. M. A. **93**:1538 (Nov. 16) 1929.

A vast majority of defects of the diaphragm discovered during life are of congenital origin. Hernias involving the stomach alone demand surgical treatment only when disturbing symptoms persist, but those involving the transverse colon should be dealt with by the two-stage operation regardless of the age of the patient. Although children stand the operation very well, the risk of shock can be reduced by the use of a mechanical respirator with intertracheal anesthesia. In the author's series of six operations on children with intestinal obstruction due to diaphragmatic hernias, those in which a two-stage operation was performed survived. This operation converted what would have been a complicated major endeavor into two relatively safe and simple procedures.

BONAR, Salt Lake City.

MORTALITY OF APPENDICITIS IN CHILDREN: A CRITICAL ANALYSIS. H. E. COE, Northwest Med. **28**:416 (Sept.) 1929.

The mortality of appendicitis in children is reported to be between 9.7 and 33 per cent, whereas the adult mortality is from 2.2 to 5 per cent. This difference is due to a number of factors, among which are the following:

1. Difficulties of diagnosis. These are often inexcusable and due to lack of patience and lack of ability on the part of the examiner. The exclusion of pneumonia, however, presents a real difficulty.

2. Delayed attention. The mother frequently fails to realize the seriousness of the situation.

3. The use of purgatives, which interferes with isolation by the formation of adhesions.

4. Conditions peculiar to childhood. The small, readily perforating appendix of the child adds to the gravity of the situation.

5. Poorly timed and badly executed operations. Immediate operation is indicated in the first two days, but often after this time, delay is productive of better results.

SCOTT, Philadelphia.

PES CAVUS: A CLINICAL STUDY WITH SPECIAL REFERENCE TO ITS ETIOLOGY. ESMÉ GILROY, Edinburgh M. J. **36**:749 (Dec.) 1929.

Gilroy discusses hollow claw-foot. In proportion to the length of the foot, the arch in infants is as high as in adults, though it may seem flat. An arch that is abnormally high in an infant is not noticeable, while the associated dorsal retraction of the toes is usually overlooked or its significance not appreciated. Many infants are 19 or 20 months of age before they try to walk. The bony deformity takes years to develop. A normal limit for the physiologic height of the arch has never been defined. The author concludes from his series of cases that if pes cavus is obscure in cause it is probably congenital, and that treatment is needed in boys at 12½ years of age and in girls at 11 years. The largest single cause is congenital; in a few cases it is hereditary. Poliomyelitis, specific fevers and posture may produce claw-foot; in fact there were twenty-four cases from such various causes, the total equaling those from congenital and hereditary causes.

NEFF, Kansas City, Mo.

ANGIOMA OF THE TONGUE. HAROLD EDWARDS, Proc. Roy. Soc. Med. **23**:38 (Nov.) 1929.

A case of angioma of the tongue was reported in a girl aged 10 years. In July, 1929, the mother noticed a few small vesicles on the dorsum of the tongue.

In two months they covered the whole tongue. Swallowing became increasingly difficult because of progressive enlargement. There was no enlargement of the cervical glands.

WILLIAMSON, New Orleans.

THE PROPER TIME FOR OPERATIONS IN CHILDHOOD. FRITZ EWALD, München. med. Wchnschr. **76**:1708 (Oct. 11) 1929.

Ewald discusses in detail the proper time for operative intervention in the many surgical conditions occurring in infancy and childhood. Young infants withstand the shock of an operation and inhalation anesthesia very poorly. If possible, rectal anesthesia should be used. It is employed in about 25 per cent of the cases in the Children's Hospital in Munich. Only malformations which seriously interfere with the infant's development should be corrected before 3 months of age and preferably after the first year.

BRAHDY, Mount Vernon, N. Y.

DEFORMITIES AND ARTHRITIS. J. VAN ASSEN JZN, Nederl. Tijdschr. v. Geneesk. **2**:4133 (Sept. 7) 1929.

Except for pelvic deformities, the significance of rachitic deformities for a later period is greatest in talipes planovalgus. Early treatment may correct or cure the unfavorable position and form of the feet. Deformities of the knees and of the spine in a number of cases also give rise to complaints in later life. Therefore it is necessary to pay attention early to these malformations and to treat the patients in time. Operation is rarely necessary. The significance of static arthritis in cases of rachitic deformities is generally overrated.

The author comes to his conclusions after observation of 1,460 patients.

VAN CREVELD, Amsterdam, Holland.

RECOGNITION OF ACUTE APPENDICITIS AND PSEUDO-APPENDICITIS IN CHILDREN. R. J. HARRENSTEIN, Nederl. Tijdschr. v. Geneesk. **2**:4612 (Oct. 5) 1929.

The author investigated to ascertain how often the diagnosis of acute appendicitis was erroneously made in a group of children. During the same period there were collected 100 successive cases in which the removed appendixes were inflamed; in 62 other cases children were sent into the hospital with the diagnosis of acute appendicitis, but in 41 of these on close observation in the hospital another diagnosis was made, or an immediate operation was not found necessary. In the remaining 21 cases which were investigated by the author himself and in which an operation for appendicitis was performed the appendix was not found to be inflamed. The involvement of the appendix found in the series of 100 cases ranged from slight to severe. In three cases the rectal temperature was below 37 C. (98.6 F.), whereas from one to two hours later at operation the appendixes showed severe symptoms of inflammation. In four of such cases the increase of temperature was only slight.

In children the presence of albumin and corpuscular elements in the urine may not be used as an argument against the diagnosis of appendicitis; neither may the occurrence of redness of the throat or of the tonsils be so considered.

In cases of pseudo-appendicitis the symptoms generally are so inconstant that they may change before the child is brought to the hospital. In the cases studied, complaints of abdominal pain and vomiting were predominant. Other symptoms more typical of appendicitis, such as rigidity and pain on pressure in the ileocecal region, often failed or were inconstant. In the greater part of the cases another diagnosis than appendicitis could be made within a few days. But in eighteen cases in which no other diagnosis could be made and in which an operation was not performed, appendicitis was highly improbable on the grounds that for more than four years after their discharge from the hospital the patients had no abdominal complaints.

The diagnosis of the twenty-one cases in which an operation was performed and in which no disease of the appendix was found are discussed in detail. In nine cases no cause for the pseudo-appendicitis could be found.

VAN CREVELD, Amsterdam, Holland.

INTUSSUSCEPTION. J. GLOS, Časop. lék. česk. **66**:55 (Jan. 7) 1927; 105 (Jan. 14) 1927.

According to Nothnagel, the invagination is due to either spasm or palsy of the circular muscle. Intussusception is more likely to be acute in children and chronic in adults. The most common site is the ileocecal region. The etiology may include sudden change in diet, as at weaning, and at times polyps, cysts or other tumors. Case reports with symptoms are cited. The differential diagnosis includes enteritis, acute gastro-enteritis, follicular colitis, appendicitis and peritonitis. The prognosis depends on the rapidity of correction. Rectal insufflation of air has been discarded in the treatment because of the possibility of rupture of the bowel. In some cases preliminary rectal instillation of warm water with elevation of the buttocks is recommended.

STULIK, Chicago.

THE CARE OF INFANTS WITH CLEFT DEFORMITIES (FACIAL). OTTO KEJŘ, Časop. lék. česk. **66**:258 (Feb. 11) 1927.

Breast milk is all important for feeding the infant with cleft deformities. Breast milk should be stimulated as vigorously as possible by manual expression, so that the infant may eventually suckle the mother's breast. Stripping and feeding with a spoon is advocated. Marchand's case is cited from the standpoint of etiology. Of fifteen children born to two mothers and the same father, nine were stillborn (all of the first mother), four had harelip (two of each mother) and one of the second mother had a "wolf face" prominent upper dental, narrow arch). These infants must be protected against infection of the upper respiratory tract. Operation is best performed when the infant is gaining, when there is no sign of infection of the upper respiratory tract and at about 2 months of age. A secondary operation is recommended at 10 months and the final one at about 2 years. The operation on the soft palate should be done before the onset of talking, to minimize the nasal twang.

STULIK, Chicago.

Miscellaneous

ROENTGENOLOGICAL CONSIDERATIONS OF THE MEDIASTINUM. WEBSTER W. BELDEN, Am. J. Roentgenol. **19**:36 (Jan.) 1928.

Conditions which must be considered in the differential diagnosis are: acute and chronic mediastinitis, mediastinal abscess, tuberculous mediastinitis, syphilitic mediastinitis, mediastinal metastases, aortic aneurysm, substernal thyroid, persistent thymus, dermoid cyst, glandular tumors, lymphomas, Hodgkin's disease and postmeasles and pertussis enlargement. The substernal thyroid shadow is usually triangular, with the base upward, while an enlarged thymus is usually keystone in shape, with the base merging with the cardiac silhouette. In swallowing, the shadow moves if it is due to goiter, and does not move if it is a mediastinal lesion. A case of persistent thymus with rib erosion before institution of treatment is cited in the discussion.

STULIK, Chicago.

CLINICAL EXCRETION OF MERCURY. H. N. COLE, JOHN A. GAMMEL, J. E. RAUSCHKOLB, NORA SCHREIBER and TORALD SOLLMANN, Arch. Dermat. & Syph. **17**:625 (May) 1928.

The authors have made a study of the urinary, fecal and total excretion of mercury in three groups of patients treated with intramuscular injections of mercuric sodium bromide (10 mg. per day), simple inunctions and inunctions with

subsequent removal of excess ointment with benzene. They have found that the variation between different patients treated by the same method is wider than the differences between the medians of the different methods. The rate of excretion increases with the duration of the administration, but at somewhat different rates. At the end of the usual three or four weeks' course, the total daily excretion averages from 1 to 1.5 mg., the extremes being 0.5 and 3.5 mg. With simple inunction it averages about one-fourth greater and with clean inunctions about one-fourth less. The ratio of mercury in the urine to that in the feces varies widely. The total amount of mercury excreted in the four weeks' course of injections averages 21 mg., of which 15 mg. is in the urine. After discontinuance of the administration, the amount excreted is high for a considerable time.

This is the second paper of a series.

SHIRLEY, Iowa City.

ULTRAVIOLET LIGHT. C. GUY LANE and ETHEL M. ROCHWOOD, Arch. Dermat. & Syph. **19**:459 (March) 1929.

Several methods for measuring the dosage of ultraviolet light are reviewed. It is argued that the "erythema dose" now commonly used is not a satisfactorily accurate standard. Although there is as yet no practical clinical method of measurement of a dose which is quick, easy of application, relatively inexpensive, and accurate for practical purposes, the most satisfactory test so far developed is the lithophone test, suggested in 1924 by Dr. Janet Clark of Johns Hopkins Hospital and developed by Dr. A. H. Pfund. The test is advocated as a satisfactory measure of the comparative efficiency of various ultraviolet lamps, and as a check on the efficiency of a given lamp from time to time. The defects of the test are that the matching of colors is a handicap and that as yet there is not a wide distribution of a stable product.

SHIRLEY, Iowa City.

HEALTH CONDITIONS OF MOTHER AND CHILD: THE PRENATAL AND POSTNATAL PERIODS. HYMAN GOLDSTEIN, Arch. Pediat. **46**:108 (Feb.) 1929.

The author makes a plea for the prenatal and postnatal care of the mothers, with especial emphasis on possible endocrine disturbance in the mother giving rise to abnormal physical or mental development of the infant. Psychic disturbances in the mother may have a marked influence on the infant. Infants should not be cared for by the obstetrician but should, when possible, be placed under the supervision of the pediatrist. The prevention and correction of physical defects and the mental training of the child are discussed. The rôle of the physician in health conservation of the child is emphasized.

WILSON, Norfolk, Va.

SIMPLICITY IN TEACHING PEDIATRICS. J. P. CROZER GRIFFITH, Arch. Pediat. **46**:312 (May) 1929.

For the unfitness of many students for bedside work, several sources may be blamed: 1. The graded courses. There should be a greater correlation between the subjects which the student has studied and their application to disease. 2. The excess of laboratory work. The writer thinks that there is absolutely, as well as relatively, too much time spent in laboratory work as compared with the time spent in bedside study. 3. The teaching of pediatrics itself. The teacher is interested in obscure and difficult conditions and is tempted to spend more time on these than on the more commonplace disorders. Teachers should emphasize the bedside management of sick children under home conditions, and the study there of symptoms, diagnosis, indications and treatment. The failure to do this is partly the fault of the teacher, partly the fault of the curriculum of the colleges, which does not stress for the student that which is actually most important for him to know. Textbooks frequently contain much which could well be omitted; the same may be said of medical journals. Many of the good

articles appearing in medical journals should be published in the excellent special journals devoted to the subjects of which they treat. WILSON, Norfolk, Va.

TABLES OF AVERAGE AND OPTIMUM WEIGHT FOR HEIGHT. WILLIAM R. P. EMERSON and FRANK A. MANNY, Arch. Pediat. **46**:382 (June) 1929.

An ideal table of average weight will not be possible until one is made from the weight and height of persons selected on a clinical basis of health diagnosis. The tables presented have been tested by use in comparison with mass and individual weights and heights of members of large groups with which the authors have worked, including in some instances the entire membership of colleges. It has long been clear that the average weight for the years of the period of growth is lower than the weight found best to serve the needs of the majority of persons. In the tables published by the authors, use has been made of the averages as the most serviceable basis for determining by clinical methods a range of optimum weights for various heights within which the person may find that weight which best meets his requirements for safety, well-being and effective living. The authors were not able to find clinically that a difference of two or three years in age at a given height necessarily called for variations in the range of optimum requirements. For that reason they have used the form of the table that gives the average weight for height without reference to age. However, the child who is habitually retarded in weight is also retarded in height. The tables show average weights and heights at various ages, the increase in weight at various ages, the average, 7 per cent the under average and the optimum weight for heights of 36 inches (91.4 cm.) and above and the average and 7 per cent below the average weights for heights from 20 (50.8 cm.) to 36 inches.

Reasons are given for consideration of the tables published. The optimum limits of weight range from 5 to 15 per cent above the average for males and from the average to 10 per cent above the average for females. This optimum range affords adequate scope for the consideration of such important factors as age, build, tendencies of a type toward fatty, bony or muscular tissue, and temperament.

WILSON, Norfolk, Va.

A REPORT ON INFANT MORTALITY. LEAGUE OF NATIONS, Pub. Health Rep. **44**: 2268 (Sept. 20) 1929.

This is the report of the conference of health experts on the protection of early infancy, the inquiry being carried out under the auspices of the League of Nations. Germany, Austria, France, Great Britain, Norway and Holland were represented. In the rural districts the mortality varied from 3 per cent in England and Norway to 10 per cent in the Netherlands, Austria and Germany. In the urban districts the mortality varied from 3 per cent in Oslo, Leyden and part of Vienna to 10 per cent in other districts of Vienna. The following measures were recommended: (*a*) education of the public as to hygiene, (*b*) education of the physicians and midwives as to hygiene and (*c*) surveillance of infants by visiting nurses.

SANFORD, Chicago.

SCHOOL VENTILATION IN CATTARAUGUS COUNTY, N. Y. T. J. DUFFIELD, Pub. Health Rep. **44**:2383 (Oct. 4) 1929.

In general, one and two room rural schools appear to be highly unsatisfactory from the standpoint of heating and ventilation. Absenteeism averaged 14.1 per cent, which is twice as high as the averages observed in the larger cities. That reported as due to respiratory infections averaged 4.7 per cent, which was twice as high as the rate for larger cities. While the incidence of respiratory infection appeared to be greater during the cold months of the year, low temperature did not appear to be directly associated with respiratory illness.

SANFORD, Chicago.

VARIATIONS IN THE KETONE BODIES OF THE URINE, BLOOD AND SPINAL FLUID IN THE DISEASES OF CHILDREN. F. PEOLA, Riv. di clin. pediat. **27**:422 (May 30) 1929.

Acetone bodies were found in slightly smaller percentage in the blood than in the urine of children showing acetonuria. Betaoxybutyric acid was found in higher percentage than acetone in both the urine and the blood.

Acetone bodies were not found in the spinal fluid in measurable quantities, except when there was some pathologic change in the choroid plexus or of the meninges, as in meningitis or meningismus.

HIGGINS, Boston.

OXYGEN IN THE TREATMENT OF CHILDREN. V. MIKALOWSKI, Wien. klin. Wchnschr. **42**:1048 (Aug. 8) 1929.

The author gives a brief historical review of the therapeutic use of oxygen given by subcutaneous injection in cases of cyanosis. He cites six cases of his own in which this treatment was given, but admits that the recovery cannot be proved to be due to this means alone. The quantities of oxygen given in these cases seem to be insufficient.

CLAUSEN, Rochester, N. Y.

POISONING BY RAW SPROUTED BEANS. H. FASCHINGBAUER and L. KOFLER, Wien. klin. Wchnschr. **42**:1069 (Aug. 15) 1929.

Two cases are reported in which after the ingestion of ten and three raw sprouted "fire beans" (*Phaseolus coccineus*) there occurred severe phasin poisoning, manifested by acute gastro-enteritis, enlargement of the liver and urobilinuria. Feeding experiments with mice and experiments with agglutination of the red blood cells of the ox and of the guinea-pig clearly showed that the phasin content of fire beans is not greater than that of the common bean (*Phaseolus vulgaris*) and that in germination, little if any phasin is destroyed. The substance is inactivated by cooking. The board bean (*Vicia fava*), eaten raw in Italy, contains much less phasin. An excellent discussion of the literature is included.

CLAUSEN, Rochester, N. Y.

THE LAZY PUPIL. P. P. BLONSKY, Ztschr. f. Kinderforsch. **36**:1 (Nov. 2) 1929.

The author distinguishes between three types of lazy pupils. The first type he most commonly finds to be children who are unusually active but who have no patience or tenaciousness. They are healthy and seek an outlet for their vigor, but have never been trained to serious endeavor. This type of child is harmed by the form of schooling which requires long hours of unbroken mental activity. The second type of laziness is found in the apathetic, weak child. It is often associated with true mental deficiency. Sometimes the physical improvement of the child is sufficient to restore the child's mental activity. The third class is composed of children who have been placed in classes which are not adapted to their stage of development; either the work is so easy that the child is not forced to put out any real energy, or it is so far beyond his capacity that he despairs.

REICHLE, Cleveland.

THE FAMILY AS AN EPISODE IN THE CHILD'S MIND. A. BUSEMANN, Ztschr. f. Kinderforsch. **36**:17 (Nov. 2) 1929.

This article is a continuation of the author's extensive and interesting investigations into the influence of the child's environment on its character and school work. It is characterized by an exceptionally judicious use of statistics and fine psychologic insight. In this publication Busemann has studied the attitude of the child toward siblings and parents; "attitude" is to be interpreted as the subjective view that the child takes and not as a veracious report of actual circumstances.

The children, all girls, were asked whether they desired more brothers or sisters or whether they were satisfied with their lot. It was found that the pupil who was the only child in the family was the most dissatisfied; the differences between these children and those in families with four and even seven children were very large. The percentages of satisfied children sank to a minimum in families of four children. Most of the children desired either a younger sister, as an object of mothering, or an older brother, as a protection or a social asset. The latter attitude was particularly prevalent in girls about the age of puberty. The pupils were again asked whether they were satisfied with their position in the family group as first, second, third, etc., child. The nearer the pupil was to being the last child, the more satisfied he was. Dissatisfaction with the family position and with the environment increased with the age of the child. In contrasting a group of boys with these girls, it was found that the girls in general were more satisfied with their lot.

The larger the family, the more satisfied the child apparently is. Busemann, of course, remarks that this satisfaction needs not be actually justified; it merely expresses the child's attitude, which can possibly be explained by several different factors: the lack of differentiation of personalities in the larger families, the social adaptation which such families encourage, and the fact that in such families a cameraderie may grow up between the siblings. Children of poorer families were apparently more satisfied with their lot. This again is apparently due to a lower standard and to a lack of differentiation, as there was certainly no more true affection in these families than in the families of wealthier persons. It is interesting in connection with the discussion of the mechanism of the "incest horror" that the older the children were, the more they tended to regard individual siblings of the opposite sex as responsible for family differences. Discussions of the theme, "What experience did you have with a member of your family?" brought out the interesting result that for the child the family was still a unit in which the individual was of less importance than the group. There was little attempt made at differentiation in the attitude or experiences of the different members.

REICHLE, Cleveland.

Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

Regular Meeting, Nov. 14, 1929

F. ELMER JOHNSON, M.D., *Presiding*

EPIDERMOLYSIS BULLOSA. DR. HOWARD REID CRAIG.

O. F., a boy, aged 26 months, whose father, aged 40, mother, aged 24, and brother, aged 4, are living and well, was presented as having a case of epidermolysis bullosa. There was no history of familial disease. There was no other case of this condition in the family, or in its collateral branches. The serologic tests of the family for syphilis were negative.

This patient was born on Sept. 28, 1927, at full term. The labor was normal, and the birth weight was 7 pounds, 2 ounces (3,231 Gm.). The child appeared to be normal. Three days after birth, there appeared large multiple bullae which had successively disappeared and reappeared up to the time of presentation. The child's skin was never entirely clear of lesions. These bullae had appeared after trauma to the skin, became large and opened. Then they became encrusted and disappeared, sometimes leaving large denuded areas and sometimes leaving mild and transient pigmentation. During periods of varying length, the patient was an inmate of various hospitals. During this entire period he remained well, grew and thrived. His appetite was good and his digestion, normal. The lesions, which often itched, made him irritable at times.

On examination, the child was well developed, moderately well nourished and not ill. It was obvious, however, that he was overly irritable and he was in some discomfort from the skin lesions. These were large and extensive bullae, involving the buccal mucosa, the hard palate, the head, the face, the extremities, the wall of the chest, the abdomen and the finger-nails and toe-nails. The contents of the bullae were serous, serosanguineous and hemorrhagic. Over the anterior portion of the scalp and the forehead there was much encrusted sebaceous-like material. By simple pressure, the skin became loosened and desquamative and was easily denuded. The upper eyelids exhibited small, pin point blebs with some desquamation along the hairline. Several patches on the face had become encrusted and covered with old coagulated blood. On the left arm was a large bulla about the size of an egg which contained blood-tinged fluid. There was a similar lesion on the left foot. The nails of the fingers and toes showed a generalized, semigangrenous desquamation. The palms and soles were not involved. There were two small bullae on the left buccal mucosa and one on the hard palate. There was a similar area on the wall of the chest.

The tonsils were greatly enlarged. There was a thick purulent nasal discharge. The cervical and inguinal lymph nodes were slightly enlarged. The heart and lungs were normal. The abdomen was soft and normal. The liver was felt at the costal border, and the spleen could not be palpated.

Successive red blood counts showed about 5,000,000 cells. The white cell count varied between 9,000 and 13,000, a normal distribution of the cells. The urine was repeatedly normal. The blood chemistry was as follows: nonprotein nitrogen, 26.5 mg. per hundred cubic centimeters; sugar, 125 mg.; chlorides, 550 mg.; calcium, 10.4 mg.; phosphorus, 4.8 mg.; albumin, 4.34 Gm. per hundred cubic centimeters, and globulin, 2.7.

The comparison of the nonprotein nitrogen and chlorides of the blood plasma and bulla serum follows: The nonprotein nitrogen in the blood plasma was

22.2 mg. per hundred cubic centimeters and in the bulla serum, 25; the chlorides, 575 and 650, respectively.

The Noguchi and Kahn tests for syphilis were negative. Cultures from the bullae showed only *Staphylococcus albus*. Successive injections of mother's whole blood, injections of solution of pituitary and ultraviolet light sufficient to produce peeling had no effect on the appearance of bullae.

This condition was first described by Goldschneider in 1882, and since then has been studied by several workers. The condition is very often congenital or familial. The etiology is still a matter of speculation. Elliot of Cornell, from studies of exceptionally clear sections, concluded that these patients suffer from congenital hyperirritability of the cutaneous nervous system and the consequent instability of the vascular system, which, when stimulated, easily pours out blood elements. Engman and Mook demonstrated a deficiency of the normal, elastic tissue fret-work of the skin. They suggested that prolongations of the terminal fibers of this elastic tissue between the cells of the rete which bind the epidermis to the corium are absent. Koebner suggested before this time, a lack of cohesion between the skin layers. Other workers ascribed the condition to various endocrinopathies and abnormal metabolic states. The most recent of these, Guy, in 1927, reported three cases in one family; but in none of these cases could he demonstrate a lack of cutaneous elastic tissue. The three cases ran in parallel courses, and Guy laid much stress on the fact that all three brothers showed roentgenologic evidence of an enlarged thymus, and during acute exacerbations, the patients showed a condition of lymphocytosis, slight generalized adenopathy and indicanuria.

In addition to presenting the patient, a few slides were shown which demonstrated various phases of the bulla production, and also two slides of photomicrographs of excised sections of skin, stained for elastic tissue. The first slide showed a section excised at the margin of a bulla. The roof of the bulla was seen to be made of the epidermal layer, including the rete. The floor of the bulla was the papillary layer. In this section, there were no obvious pathologic changes in the cells of the various layers. In the second slide, which was a higher magnification of a portion shown in the first slide, there was no evidence either of an inflammatory outpouring of blood elements, or of a lack or decrease of elastic tissue.

THE BONE LESIONS OF CONGENITAL SYPHILIS; THE CORRELATION OF THE ROENTGEN AND PATHOLOGIC OBSERVATIONS. DR. STAFFORD MCLEAN.

This article will be published in full in a later issue.

DISCUSSION

DR. RALPH S. BROMER, Philadelphia: Dr. Pendergrass and I have been much interested in the study of the bone lesions of congenital syphilis from the roentgenologist's point of view. We did not have the opportunity that Dr. McLean has had to study the pathologic specimens and slides. Our efforts were largely directed toward the attempt to arrange in roentgenologic sequence the changes in congenital syphilis and so to make it easier for the roentgenologist to recognize immediately the various stages of the disease and make him better able to cooperate with the pediatrician in making a diagnosis. We divided the changes of osteochondritis into three stages based for the most part on, and analogous to, the three pathologic stages described by Wegner.

I think that Dr. McLean has brought out a very important point in his description of his first two types of cases, group A and group B; the first group shows the early osseous lesions with a widening of the zone of temporary calcification, and the second, in addition to the wide zone of temporary calcification, shows a submetaphyseal zone or rarefaction. To a certain extent, these two stages agree with the first two stages as described by Dr. Pendergrass and myself. Both group A and group B are analogous to the first two pathologic stages described by

Wegner. It is generally admitted that congenital syphilis and scurvy show bone changes that are due to a deficient blood supply and hence we should expect to see similar roentgen changes in both diseases.

I think the lesson to be gained from Dr. McLean's paper, especially for roentgenologists, is that we have no right to say that the wide zone of temporary calcification at the end of the shaft means syphilis, just as, for instance, we cannot tell whether periosteal or cortical thickening in the middle of the shaft of a bone in older children is due to chronic osteomyelitis, syphilis or to tuberculosis of the shaft. We have to depend on other evidence to make the differential diagnosis and, as Dr. McLean has pointed out, the syphilitic lesions are often symmetrical and may show different stages of progress in the various bones. If an advanced stage is shown in one bone with merely a wide zone of temporary calcification in another, we have evidence that the widened zone is due to syphilis. And, of course, dependence must be placed on the serologic tests and other clinical signs in making the differential diagnosis.

The saw-tooth projections, as I understand them, are more prominent on the diaphyseal side of the zone of temporary calcification than on the epiphyseal side. I think everyone agrees with Dr. McLean that the saw-tooth metaphysis is a valuable sign in the diagnosis of congenital syphilis. These projections are hard to bring out in the roentgenogram, especially on the diaphyseal side, and particularly in the early stage of their formation. Perhaps we can secure a better technic in making roentgen exposures that will show these projections earlier and so make it possible to make an earlier definite diagnosis of congenital syphilis.

I feel that I am not qualified to discuss the pathologic changes of the disease. As I stated, Dr. Pendergrass' and my work was largely roentgenologic and we based our roentgen signs and stages on the work of previous pathologic workers after search and study of the literature. We noticed one particular point in the study of our cases which Dr. McLean has not touched on; that is, a lack of periosteal changes on the posterior border of the femur, even though marked periosteal change was present in the other bones. This occurred so often that it seemed to be characteristic of the syphilitic process. I should like to ask Dr. McLean if he has noted the same peculiarity in his study of periosteal lesions.

One other point is that the symmetrical lesions of the upper, inner borders of the tibias are definite roentgen signs of syphilis, but when a lesion occurs, isolated in one tibia, one should beware of making too definite a diagnosis. About a month or more ago, I did make such a diagnosis. The patient was operated on and a case of pyogenic osteomyelitis was found.

I have two slides showing lesions in the flat bones, a matter which Dr. McLean did not mention in his paper, as he stated in his introduction that he would show changes only in the long bones. The first slide, of a stillborn fetus, loaned to us by Dr. McCord of Emory University, shows clearly the double lines in the ilium and other pelvic bones and in the vertebrae. The metacarpals and the phalanges show the same changes as in the long bones to which he drew your attention. The second slide illustrates the same change in the tarsal bones. Such lines can also be seen frequently in the epiphyseal centers. In an earlier stage, the tarsal bones do not show these double lines but small areas of rarefaction contiguous to the zone of temporary calcification can be seen. They lie close to the zone of temporary calcification but do not seem actually to obliterate it.

In closing, I want to emphasize again the important point that Dr. McLean brought out in his description of his first two types of cases. We cannot diagnose every widened zone of temporary calcification that is shown in the roentgenogram as congenital syphilis. We must take into consideration the serologic evidence, the presence of other demonstrable lesions in the skeleton and other clinical signs, before a definite diagnosis can be made.

DR. EDWARDS A. PARK, Baltimore: I think that Dr. McLean has gone about this study in the right way; he has studied the bones in order to find out the cause of the shadows in the x-ray pictures. I have little sympathy with those men, who, from their arm chairs, speculate concerning shadows in x-ray plates rather

than trouble to walk to the postmortem laboratories and learn the truth by using their own hands and eyes. We shall never succeed in the interpretation of x-ray plates of bones until we compare the shadows in the plates with the anatomic structures in the bones themselves. You must now excuse the scattered nature of my remarks.

I think that Dr. McLean's reference to the correspondence between the degree of syphilitic involvement of the ends of long bones and the rapidity of growth in length at those ends is interesting. According to Schmorl, rickets appears first in the rapidly growing middle ribs, next at the lower ends of the femora and, almost at the same time, at the upper ends of the humeri and the upper and lower ends of tibiae and fibulae. The slowly growing upper ends of the radius and ulna, the lower end of the humerus and the upper end of the femur begin to show rickets only after the disease has become well developed at the fast advancing cartilage shaft junctions of the lower ends of the radius and ulna and at the ends mentioned of the other long bones. The extent of rickets at the cartilage shaft junctions of the long bones corresponds so exactly with the rate of growth at those junctions that the disease can be used as an exact means of determining the relative rates of growth at the ends of all the long bones. In a general way, in the newly born infant, not in the older infant, congenital syphilis is best developed at those cartilage shaft junctions where growth is most rapid; but the degree of syphilitic involvement does not correspond to the degree of growth with the same accuracy as in rickets.

I was interested to hear Dr. McLean state that the lower end of the ulna seemed to be the part of the skeleton which most commonly showed congenital syphilis in x-ray plates. According to the measurements of Dr. Eliot and Dr. Souther of New Haven, the lower end of the ulna grows a little more rapidly than the lower end of the radius, and in x-ray pictures, the lower end of the ulna certainly shows rickets earlier than the lower end of the radius. If the degree of rickets is the same in both, a slender bone will show the disease in the x-ray plate sooner than a thick bone. On account of the thinness, there is less superimposition of shadows, and in consequence the rachitic detail is clearer. I am also under the impression that the extension of the cortex around the proliferative cartilage, which is the first step in the development of cupping, is more apparent in x-ray plates of thin bones than of thick bones. When Dr. McLean called attention to the tufted appearance of the lower end of the ulna as characteristic of congenital syphilis in roentgenograms of the skeleton, I wondered if the thinness of the ulna as well as the rapidity of its growth and the stress and strain to which it is subject might not have something to do with the peculiar deformity which the lower end of the ulna is apt to show as seen in the x-ray plates in early congenital syphilis.

I was also interested to hear Dr. McLean point out that "osteomyelitic" lesions, developing symmetrically in the inner aspects of the upper ends of the tibiae, and giving rise to the appearance of symmetrically placed holes, are so characteristic of congenital syphilis.

Dr. McLean alluded to the fact, which I think has often been pointed out by pathologists, that the lesions of congenital syphilis may be marked in a certain tissue of the body, and not exhibit themselves in other tissues to the same extent; for example, as in the x-ray picture of the baby who showed almost no lesions of the skeleton, but veritable ravages of the disease almost everywhere in the skin.

The reverse of this case is common, namely, to find almost no evidence of syphilis on the surface of the body, but extreme development of the disease in the skeleton.

I do not think that I ever entirely understood the reason for the dense shadows at the ends of the long bones in early congenital syphilis until Dr. Dalyell of Australia, one of Miss Chick's collaborators in the Vienna post-war rickets studies, showed me at Yale one of Dr. Wimberger's histologic preparations from a case of congenital syphilis. Between the cartilage, which, in this instance, seemed fairly normal, and the older part of the shaft which showed no sign of syphilitic

involvement, there was a zone from 2 to 3 mm. in diameter that was filled with a dense lattice. This lattice was formed by interconnecting columns of the calcified ground substance of the cartilage arranged in parallel rows in the longitudinal diameter of the shaft, and separated from each other by spaces not greater than the width of one or two cartilage cells. These thickly placed parallel columns of calcified intercellular substance presented a formation that, for purposes of illustration, I might compare to a picket fence or hedge. The calcified columns corresponded to the pickets or main stems of the hedge. Now, these columns of calcified intercellular substance were nowhere encased in bone, or, if encased at all, only by thin films in the parts bordering on the normal shaft. Moreover, they were everywhere present and intact. The microscope made clear what had taken place in the bone. The growth of the cartilage had continued while the syphilitic infection was in operation, so that the bone had increased in length and the cartilage, true to its function, had kept on forming and leaving behind, as it grew, calcified intercellular substance, arranged in orderly columns which reflected the orderly arrangement of the cartilage. The shaft had continued as usual to remove the ripe cartilage cells, but had lost its power to form bone on the columns of calcified ground substance of the cartilage, with the result that these columns simply persisted in this zone without encasements of bone. In healthy, growing bone, the columns of calcified intercellular substance, continuously being left behind by the cartilage in the course of its growth, survive only occasionally. The great majority of the columns are probably not needed for mechanical purposes and are at once or soon removed by the cells which the body uses when it is necessary to destroy calcified cartilage or calcified bone. It is to this regular, orderly destruction of calcified intercellular substance and trabeculae that the marrow cavity owes its formation. In the specimen brought by Dr. Dalyell, the body had evidently lost its power to destroy those columns of calcified cartilaginous ground substance, which, under normal circumstances, would have been destroyed. The disease had, therefore, altered normal processes in at least two ways; it had inhibited the orderly activity of the osteoblasts, so that no bone formation could take place on the surface of the columns of calcified intercellular substance and it had inhibited what might be termed the physiologic destruction of the newly formed, calcified, cartilaginous, intercellular substance and bone just under the cartilage. The result was the formation between the cartilage and the healthy shaft of this fence of calcified, cartilaginous, intercellular substance, at all points unbroken and lacking bone encasement. We know from the studies of Weech that the dense shadows which appear in the x-ray pictures of bones are cast only by deposits of lime salts. It is entirely understandable, then, why such a bone as the one described should have cast a dense, bandlike shadow at the end of the shaft immediately underlying the cartilage on the x-ray plate.

Now, usually congenital syphilis does not affect the growing ends of the long bones in this easily understandable way. It is, of course, always true that when the cartilage shaft junction of a rapidly growing bone is involved in syphilis, osteoblastic activity is suspended or diminished so that the calcified columns of ground substance do not receive encasements of bone; it is also true that the physiologic destruction of the newly formed calcified columns is inhibited. But a third factor almost always enters in sooner or later, especially when the disease is severe and has been of considerable duration, namely, connective tissue, which usually has some of the histologic features of syphilis, grows lawlessly in various places in the newly formed end of the shaft, where the columns of calcified intercellular substance lie, and often destroys, or at least replaces, them in considerable areas. These connective tissue growths tend to develop around the larger vessels, whether these enter the subcartilage regions of the shaft from the cartilage itself, from the periosteum or from the shaft, and they appear to be responsible for breaking up the orderly arrangement of the columns of calcified substance.

In these cases, changes in the proliferating cartilage cells also occur. When connective tissue (syphilitic granulation tissue) runs riot at the end of the shaft, the team play of the cartilage and the shaft, which is essential to normal growth,

becomes disorganized. Presumably, the circulation is interfered with, and the mature cartilage cells do not meet the capillaries in all places and they are not assimilated or removed by the capillaries as they are under normal conditions. At certain places, therefore, along the zone of proliferating cartilage, the cartilage cells accumulate and undergo degenerative changes, or at least become abnormal in a morphologic sense. They are no longer disposed in an orderly manner in parallel columns; they take on irregular shapes. I wish to emphasize especially that they produce excessive amounts of intercellular substance. Under normal conditions, the network of cartilaginous, intercellular substance produced by the cells nearest the shaft is delicate and regular in arrangement. In congenital syphilis the network formed by the intercellular substance becomes very thick in those places where the proliferative cartilage has accumulated because it was not taken up by the shaft and, since the arrangement of the cartilage is irregular, the network becomes irregular. This thick, malformed network affords more room for lime salt deposition than there is under normal conditions, and hence the deposits of lime salt into this cartilaginous matrix are copious. Another result of the failure of the shaft to absorb the cartilage in an orderly way is that the proliferative zone of cartilage, taken as a whole, becomes most irregular. The sharp linear division between cartilage and shaft disappears, and at those points where the cartilage has not been taken up by the shaft, the proliferative zone appears to be prolonged into the shaft; at other points, where masses of cartilage have been eroded by blood vessels surrounded by syphilitic granulation tissue, the shaft seems to be prolonged into the cartilage. In consequence of this unequal removal of the mature cartilage, the shaft and cartilage interdigitate, somewhat the same as in rickets, although not usually to the same degree as in advanced rickets. The practical importance of these changes, as far as the interpretation of x-ray plates in congenital syphilis is concerned, is twofold. As the result of the interdigitation between shaft and cartilage, the end of the shaft is most irregular and in the x-ray picture has a saw-tooth or serrated appearance. Because of the abundance of the deposit of lime salt in the deformed cartilaginous ground substance, the shadows cast by the deposits are especially intense.

Finally, I must call attention to the great developments of connective tissue (syphilitic granulation tissue) about the blood vessels which pass from the resting zone of cartilage, through the proliferative zone, to the shaft, the cartilage canals. Even in the normal bone one occasionally sees deposits of lime salt extending into the proliferative cartilage along the cartilage canals. In these severe cases of congenital syphilis, this phenomenon is greatly exaggerated and the deposits of lime salt sometimes extend along these mammoth diseased cartilage canals and continue completely through the proliferative cartilage. When these deposits occur along the cartilage canals in congenital syphilis, of course they cast shadows in the x-ray plates which can be seen as threadlike or flame-shaped prolongations from the end of the shaft. They add to the serrated or tufted appearance of the end of the shaft and may be mainly responsible for it. Furthermore, in these severe cases of congenital syphilis, the bone is apt to be greatly weakened at the cartilage shaft junction, and the calcified cortex becomes prolonged beyond the end of the shaft so that it embraces, or at least extends along the side of, the proliferative cartilage. The deposits of lime salts at these points give the appearance in x-ray pictures of spurs projecting from the end of the bone in the line of the cortex.

To summarize: 1. In congenital syphilis osteoblastic activity is interfered with or completely suspended, so that the calcified trabeculae or cartilaginous intercellular substance do not normally become encased in bone. This process in itself is not responsible for excessive shadow production in x-ray plates, although it is highly characteristic of the disease. 2. Hand in hand with this failure of osteoblastic activity, the physiologic destruction of trabeculae and calcified intercellular substance is suspended, so that the densely placed columns of calcified intercellular substance remain in the shaft, just as they were left by the retiring cartilage. The columns are separated only by the width of one or two cartilage cells and

hence make a dense fencelike or hedgelike formation of calcified material which, as the result of superimposition, casts on the x-ray plate a solid shadow at the very end of the shaft. These failures in osteoblastic activity and in the physiologic destruction of the newly formed trabeculae are always in evidence at the end of a bone in syphilitic osteochondritis but are rarely the only changes present. 3. Usually a third factor is added, in the form of the development of great quantities of connective tissue (syphilitic granulation tissue) at the growing end of the bone just under the cartilage. In very severe cases, these growths of syphilitic granulation tissue are invariably present and are responsible for the irregularities in the x-ray picture of the cartilage shaft junction. These growths of connective tissue are usually evenly distributed. Where they are present, the calcified material disappears; where they do not occur, one often finds dense masses of calcified material. They interfere with the interplay between cartilage and shaft with the result that the growth of the cartilage becomes most irregular and the end of the shaft becomes irregular, and, as seen in the x-ray picture, has a saw-tooth appearance. This saw-tooth appearance is further increased by the extensions of the calcified cortex around the sides of the proliferative cartilage and the deposits of lime salts along the abnormal cartilage canals. The syphilitic granulation tissue casts no shadow, and the calcified intercellular substance of the deformed cartilage casts an excessively heavy shadow. As the two may exist side by side, it is common to see in the x-ray plate, at the end of the shaft in cases of congenital syphilis, shadows of great density intermingled with shadows of almost no density.

On the basis of our knowledge of the way in which congenital syphilis affects the bone, it is possible to understand easily the peculiar shadows at the ends of the long bones. Sometimes the end of the shaft of the long bone is marked by a dense horizontal bandlike shadow. The explanation of this shadow is that the columns of calcified intercellular substance which have been steadily formed by the cartilage in the process of growth are still all present immediately under the cartilage at the end of the shaft, having neither undergone physiologic destruction nor received an encasement of bone. The density of the shadow is due to the fact that the columns of calcified intercellular substance lie close together in a thick, picket fence or hedgelike formation and are rich in lime salts.

If the band at the end of the bone is serrated, as Dr. McLean pointed out in some of the lantern slides, the meaning is that syphilitic granulation tissue has developed around the cartilage canals and immediately under the proliferating cartilage, and has interfered with the orderly growth of the cartilage and the process of calcification; and the lime-containing elements of the end of the shaft have become irregular and cast an irregular shadow. The prolongations of deposits of lime salt along the cartilage canals, and along the sides of the proliferating cartilage, are often clearly visible.

If the end of the shaft casts dense shadows at some points and at other points, no shadows at all, the explanation is that at some points, calcified, probably very heavily calcified, cartilaginous ground substance remains (the cause of dense shadow) and at the other points there is only granulation tissue or granulation tissue with a few thinned or fragmented calcified trabeculae. The wormeaten appearance of the end of the shaft means nothing more than an irregular intermingling of syphilitic granulation tissue on the one hand, and an intact network of heavily calcified cartilaginous intercellular substance and bone on the other. Perhaps it is justifiable to think of the syphilitic granulation tissue as playing the part of the worms, and of the calcified material as the substance eaten. The wormeaten appearance, then, means that the calcified material at the end of the shaft has been extensively eaten away, but that parts still remain, and these parts cast the shadows.

The rarefied zone immediately underlying the zone of increased density at the end of the shaft does not always exist in congenital syphilis. When present, as is usually the case, it can be explained on the ground that the columns of calcified intercellular substance and often the older trabeculae of bone themselves have been largely destroyed as the result of the development of the syphilitic

granulation tissue. In advanced cases of syphilitic osteochondritis one finds syphilitic granulation tissue everywhere in the part of the bone corresponding to the zone of rarefaction, and only a few remaining trabeculae or fragments of calcified ground substance. In one case, the deep zone of rarefaction at the end of the shaft was composed entirely of syphilitic granulation tissue and only a few trabeculae or fragments of calcified ground substance remained. All matter containing lime salt was gone excepting a little just under the cartilage. The cartilage itself was heavily calcified. In certain early cases of congenital syphilis, as Dr. McLean has shown, the only evidence of the disease may be a zone of rarefaction immediately underlying the normal or nearly normal shadows cast by the provisional zone of calcification of the cartilage and its immediately subjacent trabeculae at the end of the bone.

On examining some of Dr. McLean's specimens showing this early lesion in the x-ray film, I was surprised to see that the rarefaction was due solely to a diminished number and increased thinness of the trabeculae in the rarefied area. The marrow in which the trabeculae lay seemed to be entirely normal and showed no evidence of syphilis. Dr. McLean makes the ingenious explanation that the rarefaction is the result of the effect of the disease on the nutrition of the infant as a whole, the newly formed portion of the end of the shaft, having felt the emaciating influence. I should prefer, however, to still hold the view that in these cases, osteoblastic activity has been interfered with in some specific way as the result of the disease, and that the thinness of the trabeculae and their diminished number are the consequences.

The explanations which I have attempted of the pathologic process in congenital syphilis make it possible to understand why in that disease bones fracture so easily just under the cartilage through the freshly formed shaft. Osteoblastic activity is suppressed, and the columns of calcified intercellular substance, uncoated with bone, offer the resistance of china or chalk. Moreover, in severe syphilis, the extreme end of the shaft is weakened further as a result of the focal destruction of the calcified intercellular substance by the so-called syphilitic granulation tissue. It is difficult in the autopsy room to cut the bones of infants who show syphilitic osteochondritis without producing fractures through the shaft just underneath the cartilage.

A common mistake is to think that in a case of congenital syphilis a hole in the shaft of a bone, as seen in the x-ray plate, denotes the presence of a gumma. Small gummas may be present in the shafts of the bones of infants who have congenital syphilis, but the holes seen in the x-ray plates are due to the extensive foci of destruction of the lime-containing portions of the bone and the substitution of syphilitic granulation tissue.

Intensification of the shadows at the ends of the shafts of long bones is seen in conditions other than congenital syphilis. For example, the shadow at the end of the shafts of the long bones is denser in children more than 1 year old than in infants; the shadow at the end of the radius in a 2 year old child would be abnormal in a baby of 3 months. When growth stops temporarily at the end of the long bones, the shadow at the end may become intensified and seems abnormal. This is not always the case, but is the case when, as a result of a growth stasis, a transverse line is in the process of formation at the cartilage shaft junction. An increased density of the outline of the end of the shaft is characteristic of rickets in an early stage of the healing process, and also of scurvy. I think in scurvy that the intensification of the shadow is often more apparent than real, the shadow seeming intense in contrast to the lack of density in the rest of the bone. Indeed, when the skeleton is rarefied, which is often the case in wasting illnesses, the ends of the long bones seem to be marked by dense shadows, which are in reality dense only by contrast. A dense shadow at the end of the bone can be produced by the administration of phosphorus, which was originally shown by Phemister and confirmed by Hess. We have found that a dense band across the end of the shaft of the long bones can be produced in lead poisoning. We have seen intense bands, from 2 to 4 or 5 mm. in diameter,

marking the ends of the long bones in three cases of chronic lead poisoning. We do not know as yet whether the shadow is cast by the metal in the newly formed trabeculae (in the way in which Aub and his collaborators think occurs in lead poisoning), or whether, as in the case of phosphorus, the formation of trabeculae has been brought about by the lead, and the shadow is caused by a more dense formation of bone. Perhaps other heavy metals will produce intensification of shadows at the growing ends of long bones.

It is true that cupping occurs less commonly in congenital syphilis than in rickets. But the formation of spurs which prolong the line of the cortex at the ends of the bone is very common in that disease. Often a spur is present in the x-ray plate, at one or both sides of the end of a long bone, but the end of the bone continues to present a normal flat contour in contrast to the curved contour seen in advanced rickets.

If time were not lacking, I should like to discuss the differential diagnosis between healing rickets and scurvy and congenital syphilis. It is often exceedingly difficult to be certain from the x-ray plate from which of these three diseases the infant suffers, and one is often forced to rely on knowledge other than that furnished by the x-ray plate to establish a diagnosis of congenital syphilis.

PHILADELPHIA PEDIATRIC SOCIETY

JOHN D. DONNELLY, M.D., *Reporter*

Regular Meeting, Dec. 10, 1929

RALPH M. TYSON, M.D., *President, in the Chair*

CONTINUOUS TEMPERATURE RECORDS FROM NEW-BORN INFANTS UNDER EXPERIMENTAL CONDITIONS. Presented by DR. JOSEPH STOKES, JR., DR. ANGUS MCBRYDE and CHARLES TATNALL, A.B.

Continuous temperature records were presented of normal new-born infants during the first two weeks of life. Originally, an attempt was made to obtain accurate records by means of a woven nickel wire resistance thermometer, adapted for recording skin temperatures by R. M. Kreinberg. The marked fluctuations of the record with consequent lack of accuracy in determining the temperature of the infant forced the presenters to change to a thermometer which could be inserted into the rectum or vagina. This thermometer, while slightly less sensitive to changes of temperature, gave more constant variations which were comparable with the actual temperature variations of the infant.

Eighteen charts were presented showing the continuous vaginal temperatures of several female infants, with the changes consequent on changes of environment. The first charts compared the continuous vaginal temperature and the axillary temperature recorded by two instrumental recorders on the same infant at the same time. The even variations of the vaginal temperature contrasted sharply with the marked fluctuation of the skin temperature. The other charts in order showed: (1) the lack of diurnal variation in the temperature, (2) the slow but definite effect of changes in room temperature, (3) the slight effect of exposure for ten minutes to the air at 75 degrees and the effect of crying on such exposure, (4) the lack of effect of exposure to an oil bath, (5) the moderate effect of exposure to a water bath for varying intervals, (6) the usual effect of defecation and urination in lowering the temperature, (7) the marked effect of exposure to a 30 per cent alcohol bath at a temperature of 75 degrees and (8) the effect on inanition fever of exposure to a 30 per cent alcohol bath at a temperature of 75 degrees.

The preceding paragraphs constitute a preliminary report on the experimental work covered under the title.

AN AUTOMATIC RECORDING TEMPERATURE CONTROLLER FOR PREMATURE INFANTS. Presented by DR. RALPH M. TYSON and DR. EDWARD F. BURT.

This article appeared in full in the February issue of the ARCHIVES, page 321.

DISCUSSION

DR. HENRY C. BAZETT: A veterinarian first started work on temperature control in cattle twelve and a half years ago, and it has taken up until this time for its application to man. The application of control of temperature in a child has considerable importance. In my mind, the recording type of thermometer will revolutionize medicine; it will be more useful in ten or twelve years from now than the electrocardiograph as it has a much more general application to a larger number of patients. It is important to realize that the thing one sees in a child can be duplicated in the adult.

An ordinary swim in the sea will cause one's temperature to drop a couple degrees and not return to the previous level for four or five hours. The rise in temperature which is noted in a child on crying may be noted in an adult when he is in a rage. The diurnal variations, of course, have been known. Some of the observations are merely exaggerations of the normal, but we do not know enough about the normal. It seems logical to start with children and work up to the adult. The vaginal thermometer is ideal for the female. For the male, the rectal thermometer is ideal with the system which Dr. Stokes has described, which gives good control. Whether a rectal thermometer can be carried for a matter of days or weeks is another story which can be determined only by testing. The surface gives much more fluctuation. The skin warms much more slowly, and deeper control would be more satisfactory.

Temperature control should be within the reach of every ordinary private family; it has its possibilities. As for its use in the hospital, it is not so much the treatment that counts, but knowing more about the effect of a cold sponge on temperature and ultimately learning whether we know which way we want to control temperature; we do not know at present.

A STUDY OF THE INCIDENCE OF THE CAUSES OF FEVER AMONG HOSPITAL PEDIATRIC PATIENTS. Presented by DR. JOHN D. DONNELLY.

Realizing the difficulties of early diagnoses of febrile diseases, in an effort to determine the frequency of the various causes, a study was made of all the patients with fever admitted to the medical services of The Children's Hospital of Philadelphia, between the years 1919 and 1927. Three thousand eight hundred and twenty-eight cases of fever were studied as to their etiology. When possible, the diagnoses were checked with the laboratory or postmortem observations or both. No case was studied in which the patient did not have a temperature of 101 degrees or higher, continuously or intermittently for forty-eight hours or more. Those who did not have completed studies to warrant a diagnosis were not included in this study. Complications capable in themselves of causing fever, occurring while the primary disease existed, were not included; however, complications and secondary infections which occurred after the primary disease ceased to exist were included. As this study was made among hospital patients, many causes of fever of short duration, even though high, that are commonly met in private practice were necessarily absent.

In this study 66.2 per cent of all the patients were under 3 years of age. No child was more than 12 years. The striking feature of this analysis was that 48.8 per cent of all cases of fever were due to nontuberculous infections of the lower respiratory tract. If diseases of the upper respiratory tract were added to this group, then respiratory diseases would have accounted for the presence of fever in more than half of all the patients. Pneumonia composed 80 per cent of the nontuberculous causes of fever in cases of disease of the lower respiratory tract. There was marked seasonal variation in the incidence of bronchitis and

lobar and bronchopneumonia. Otitis media was next in frequency, followed by the gastro-intestinal disorders. Brain and meningeal conditions, exclusive of those occurring in the new-born infant, ranked fourth as a cause of fever. Tuberculous meningitis accounted for 44.4 per cent of the brain and meningeal conditions. Contagious diseases ranked next, followed by tuberculosis, the miliary type being the most common: the majority of the patients in this group were less than 3 years of age. A little more than 20 per cent of the miliary cases presented no evidence of meningitis while in the hospital.

Charts were available showing the grouping and incidence of the causes of fever among this large number of pediatric patients. It was found that respiratory diseases, exclusive of tuberculosis, ear infections and gastro-intestinal disorders, composed three fourths of all the causes of fever. If meningitis, contagious diseases, pyelitis, cardiac diseases and skin infections were added, one found that in more than 90 per cent of all febrile cases the diagnosis could be made with a minimum of laboratory work. With persistent effort, most symptoms will show themselves if diligently sought by repeated physical examinations, supported by practical laboratory work. However, there are times when all available skill will fail to determine the cause of fever in a particular patient. There were thirty-four patients in this large series, fourteen of whom had convulsions, in whose cases diagnoses were uncertain or were not made.

DISCUSSION

DR. JOHN F. SINCLAIR: I should like to ask Dr. Donnelly whether he found any cases of syphilis in his study which it seemed plausible to believe might have caused a rise in temperature. We have been interested in a case which has been carefully studied and in which an elevation of temperature existed for which we could find no cause. The patient had a four plus Wassermann reaction and after the institution of antisyphilitic treatment, the temperature became normal. We should like to know whether we had the cause or whether it was only associated.

DR. JOHN R. DONNELLY: There were seven cases similar to Dr. Sinclair's case, in which no other cause than syphilis was found nor any evidence suggestive of another cause of fever. The change in temperature was more or less intermittent: it fell shortly after the beginning of treatment and remained normal. There was one exception. One of these children later developed meningitis and died.

CLEVELAND ACADEMY OF MEDICINE, SECTION ON PEDIATRICS

Annual Meeting, Dec. 11, 1929

C. W. WYCOFF, M.D., *Chairman*

THE NEWER ASPECTS OF VIOSTEROL. DR. ALFRED HESS.

Standardization of the potency of viosterol is of great importance and Dr. Hess made comparison with the potency of cod liver oil. The American standard, 100-D, indicates a potency 100 times as great as that of cod liver oil. The difference in the potencies of the original preparations depends on the intensity of the irradiated solvents. Estimate of the degree of potency based on the number of milligrams of viosterol is unreliable: for instance, the 40-D original preparation of France contained 10 mg. and the 2500-D preparation of Germany contained only 10 mg. On biologic tests, it is indicated that the dosage is too low rather than too high. Fortified cod liver oil is cod liver oil to which viosterol has been added, resulting in a mixture with a potency of 5-D.

Ergosterol, while found elsewhere in nature, may be secured easily from yeast on account of its cheapness. Irradiated milk, dry or fluid, can be used in place

of ergosterol. There is not much gain in irradiating cereals, for two reasons: there is little sterol in cereal to be activated; the most serious time for rachitis in an infant is the first six months of life, during which period an infant receives little or no cereal. Of risk with many irradiated foods, Dr. Hess thinks there is little danger.

This new drug cures patients with craniotabes while in certain instances cod liver oil has failed. It stimulates increased production of eggs in fish in captivity. In rickets, as a result of treatment, hypercalcification of the epiphyses is seen in the roentgenogram.

True clinical indications for the use of this drug are seen in rickets, tetany and osteomalacia; the last disease is seen frequently in India and China. This substance has been tried in Paget's disease, dental caries, fractures, paralysis agitans and colds, in fact, as a cure all.

The toxicity of viosterol lies in its ability to produce hypercalcemia, but it is not toxic in its various chemical phases. The final product is a mixture, not a photochemical entity. As a result of hypercalcemia, there occurs calcification of the aorta, coronaries, heart, stomach and kidneys. But hypercalcemia is not distinctive of viosterol. Cod liver oil can produce the same result in rats in doses of 300 mg. and in infants in doses of from 3 to 6 teaspoons a day. There is no risk in giving viosterol in the summer months in spite of sun irradiation to the skin. Fortunately, in both animals and the human being, a definite clinical symptom occurs when hypercalcemia is imminent. Loss of appetite is noticed. This symptom gives warning in ample time before damage can occur. These toxic effects were associated with the old vigantol, the potency of which was 200 times that of our 100-D preparations. The margin of safety is very wide with the latter. In Dr. Hess' opinion, the toxic dose is at least 300 times that of the prophylactic or even curative dose.

The therapeutic dose is from 8 to 10 drops a day; 15 drops to cure patients who have rachitis and tetany, and larger amounts to protect premature infants. It must be remembered that idiosyncrasy for the substance occurs, but failing appetite as a gage of overdosage is adequate. Viosterol may safely be given to pregnant women. If there is a negative calcium balance, the woman will not be robbed of her ability to provide the infant with calcium. For such purpose, 20 drops may be given, the dose being divided between the two, but the fetus deriving the greatest benefit.

Book Reviews

THE NURSING OF INFECTIOUS DISEASES. By J. J. WOOLLACOTT, M.A., M.D., B.Ch.Oxon., D.P.H. Fourth edition, revised and enlarged by DOROTHY C. HARE, C.B.E., M.D., M.R.C.P., D.P.H. Assistant Physician Royal Free Hospital, England. Price, $1.50. Pp. 195. New York: G. P. Putnam's Sons, 1930.

The first three chapters deal with the nature and methods of the spread of infection, preventive measures and the general management of infectious diseases. The common communicable diseases are considered in the remaining fifteen chapters.

Typical temperature charts and a table of reference for the periods of incubation, etc., of the various infectious diseases are given.

It is claimed to be "a practical commonsense volume dealing with the proper methods of nursing infectious diseases. It stresses particularly the adequate care of the patient with the maximum comfort to him and a minimum of danger to others. A complete description of the chief infectious diseases and of the measures, both preventative and curative, usually taken in dealing with them is included."

It is evident from the beginning that the teaching of nursing of communicable disease is based on different concepts from those held in those hospitals in the United States, in which aseptic nursing is practiced.

In tracing the spread of infection from the sick to the well too little emphasis is placed on the rôle played by the human carrier, by fresh moist discharges and by contaminated hands. The statement is made that in some of these diseases "the virus is given off more or less from all parts of the body and becomes diffused throughout the room." I know of no disease in which there is a scientific basis for such a statement. Cleanliness of the patient and of his surroundings, the prompt disposal of his secretions by burning, of his excretions and bath water by disinfection and of his dishes, linen, etc. by boiling, will prevent the spread of disease to others if those in attendance carry out intelligent aseptic measures at all times.

In caring for infectious disease in the home the authors advocate the use of a sheet wet with a disinfectant solution hung over the door of the room occupied by the patient. Since it is known that "germs of disease do not fly, hop, walk or run" but that they are spread about by unclean hands, by contaminated things and by improper disposal of secretions and excretions, I believe that the prevention of their spread depends on an intelligent understanding and application of these facts. When the attendant understands clearly why she must always put on a coverall gown when entering the patient's room and why she must always cleanse her hands thoroughly with soap and water and remove her gown without contaminating the clean side of it or her hands before leaving the isolation area, and why no article can be removed from the room until it is prepared for burning, boiling or proper sterilization, we may expect her to carry out these procedures on a sound basis. Wet sheets hanging over doors and disinfectant solutions for hands are spectacular but dangerous, because they give a false sense of security and tend to minimize the importance of mechanical cleanliness which is a vital factor in the prevention of the spread of disease.

The danger of human carriers should not be overlooked. Cultures should be made from all members of the household and the attendant, as well as the patient, before the termination of quarantine in order to detect possible "carriers" or mild, missed cases. These matters are of far greater significance than fumigation.

The authors state that with a highly trained staff it may be possible to rely on asepsis in the management of these diseases in communicable disease hospital wards. This has long passed the experimental stage in this country even in institutions in which bedside care is given by affiliating student nurses who are in the service for only a few weeks.

At the termination of isolation we believe that boiling of dishes, utensils, linen, etc., burning of books, papers, toys and things of little value, thorough sunning and airing of mattresses and pillows if they have been properly protected from discharges and washing of furniture, floors, woodwork, walls, blankets, etc., with soap and water, remove all possible infectious material and make the room perfectly safe without carbolizing the furniture or fumigating the room as the authors teach.

The statement is made that from smallpox alone among the infectious diseases is it possible for the nurse to be protected. For many years nurses have been actively immunized against typhoid fever, diphtheria and scarlet fever just as successfully as against smallpox.

Disinfectant gargles, nasal sprays and douches are advised for nurses caring for patients with this class of diseases. I believe that such measures lower the resistance of the mucous membranes and inhibit their normal bactericidal power. It would seem much better to teach the nurse how to avoid the danger of becoming infected by fresh, moist discharges, to keep her hands away from her face, to wear a simple mask over her mouth and nose while cleansing a mouth or taking a culture from a throat and to avoid getting in line with the patient's mouth when he is coughing, crying, vomiting, etc.

In the treatment for scarlet fever no mention is made of the antitoxin which if given early checks the course of the disease and prevents the secondary complications which are often serious or fatal. Desquamation is no longer considered of any significance so far as infectivity is concerned, but the patient is infectious until hemolizing streptococcus organisms have disappeared from the nose, throat or discharging areas.

In the treatment for diphtheria the usual dosage of antitoxin given is from 6,000 to 12,000 units, with the maximum limit 60,000. In this country the average dose is about 25,000, while late cases often receive 100,000 or more. Physicians are taught to give antitoxin deeply into a large muscle which is rich in blood supply in order that the serum may be taken up into the circulation quickly, while the authors give it subcutaneously into the abdominal wall or into the loin.

In the treatment for laryngeal obstruction in diphtheria, tracheotomy is the usual procedure, while in this country, especially in hospital practice, where a medical attendant is always available, intubation is done if possible as this appears to offer better chances for an uncomplicated recovery.

The Schick and Dick tests for determining susceptibility to diphtheria and scarlet fever are referred to, but the authors state that active immunization is in the experimental stage. In this country the results obtained in many cities from the use of toxin-antitoxin or toxoid have been most gratifying, cutting down the incidence of diphtheria in a most spectacular way. In certain institutions here the Dick toxin has been used for immunization against scarlet fever for six years. The results obtained from this procedure have been equally promising and effective.

In presenting the subject of measles, mention is not made of the possibility of the use of convalescent human measles serum as a prophylactic measure. This measure, which has been in use for fifteen years, is a significant advance in preventive medicine in the treatment of the infant or the sick child who has been exposed to a disease the complications of which are fatal in such children.

The difficulty in differential diagnosis between German measles and scarlet fever is stressed. This often may be cleared up in a few hours by the simple scarlet fever blanching test, which is valuable also in the differentiation between food or drug rashes which may closely resemble scarlet fever.

For the comfort and support of children suffering from whooping cough, smooth, snugly fitting, abdominal binders are not mentioned. These binders provide mechanical support and hernias are less likely to develop when they are kept on throughout the paroxysmal stage.

Following poliomyelitis we are convinced that normal posture and absolute rest of the back as well as of the paralyzed members should be maintained until after all tenderness disappears and that exercises should be gradually increased, soreness and fatigue being avoided, and that every patient should be under the direct management of an orthopedic surgeon until function is restored to as nearly normal as possible.

It is regrettable that in the recent revision of this work the material has not been brought up to date for there is throughout the book a fine appeal to the true-spirit of nursing. The comfort and well being of the patient are always uppermost. There is much of stimulation to high ideals of service and devotion to duty which is highly commendable.

GYNAECOLOGY FOR NURSES AND GYNAECOLOGICAL NURSING. By COMYNS BERKELEY. Price, $2.75. Pp. 416, with illustrations. New York: G. P. Putnam's Sons, 1930.

This book is well written in a clear and concise manner and the text is liberally supplemented with excellent illustrations both photographic and diagrammatic. The material covers completely the fields of obstetrics and gynecology, and the author makes every effort to make the book practical. The chapters on anatomy bring this out in an effective manner. In this part of the volume each of the peculiarities of the anatomic make-up is described in terms of importance to the nurse in caring for the patient.

The book is divided into six parts: anatomy, physiology, pregnancy, parturition, diseases of the reproductive tract and gynecologic nursing. In the chapters on diseases of the reproductive tract, the congenital anomalies, postpartum relaxations, infections and new growths are exhaustively and lucidly covered. Chapters on infection, immunity and septicemia are especially worth mention.

The entire book is well worth reading and should make a splendid text for classes for nurses.

A TEXTBOOK OF ORTHOPAEDIC NURSING. By EVELYN G. PEARCE. Price, $1.75. Pp. 155, with 44 illustrations. New York: G. P. Putnam's Sons, 1930.

The first textbook on orthopedic nursing supplies the fundamental information that is necessary for nurses. With this understanding of common orthopedic conditions, the nurses are better qualified for cooperation with the orthopedic surgeon in the prevention of development of deformities, the treatment for them and the training of the patient.

The subject matter is presented in simple language and is well arranged. The clinical pictures are a great asset.

The fundamental nursing technic is explained for each condition; however, the nursing details must vary according to the surgeon.

If any criticism is to be given, I should say that the text does not include enough specific detail of instructions for nurses in the care of orthopedic patients.

CHILDHOOD TYPE OF TUBERCULOSIS. By HENRY D. CHADWICK and F. MAURICE MCPHEDRAN. Pp. 29, with 1 plate and 6 photographs. New York: National Tuberculosis Association, 1930.

Within its twenty-nine pages this manual contains more compact facts than many textbooks on childhood tuberculosis. It is well organized and well written, and is divided into sections on diagnosis and treatment of childhood tuberculosis, classification and diagnosis. The latter section includes reproductions of x-ray plates and color plates of the tuberculin reaction. This manual is available through affiliated state and local tuberculosis associations, and should be read by every physician.

SCRITTI MEDICI DEDICATI A CARLO COMBA NEL XXV DEL SUO INSEGNAMENTO E PUBBLICATI PER CURA DELLA "RIVISTA DI CLINICA PEDIATRIA." Florence: Luigi Niccolai, 1929.

This large volume consists of eighty papers, many of which had previously been reported in various journals. It is beautifully gotten up and contains most interesting material. The articles presented represent collaboration not only of the various Italian faculties but of the French, German and Hungarian physicians as well. It is a well deserved tribute to a most distinguished Italian confrère.

Directory of Pediatric Societies

INTERNATIONAL

International Pediatric Congress

Prof. I. Jundell, Artillerigatan 23, Stockholm, Sweden.
Place: Stockholm. Time: Aug. 17-20, 1930.

NATIONAL

American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman: Dr. William Weston, 1231 Bull. St., Columbia, S. C.
Secretary: Dr. C. A. Aldrich, 545 Lincoln Ave., Winnetka, Ill.
Place: Detroit. Time: June 23-27, 1930.

American Child Health Association

President: Hon. Herbert Hoover, 370 7th Ave., New York.
Secretary: Dr. Philip Van Ingen, 370 7th Ave., New York.

American Pediatric Society

President: Dr. Joseph Brennemann, 30 N. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Place: Windsor Hotel, Montreal. Time: June 17-19, 1930.

Association of American Teachers of the Diseases of Children

President: Dr. Lawrence T. Royster, State Board of Health, University, Va.
Secretary: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Place: Detroit. Time: June 24, 1930.

Children's Hospital Association of America

President: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Secretary-Treasurer: Miss Bena M. Henderson, Milwaukee Children's Hospital, Milwaukee.
Place: New Orleans, La. Time: October, 1930.

Canadian Society for the Study of Diseases of Children

President: Dr. E. A. Morgan, 160 Bloor St., Toronto, Ont.
Secretary-Treasurer: Dr. George E. Smith, 244 Bloor St., W., Toronto, Ont.
Place: Brockville, Ont. Time: June 20-21, 1930.

SECTIONAL

Central States Pediatric Society

President: Dr. Edward Clay Mitchell, 1073 Madison Ave., Memphis, Tenn.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Blvd., Chicago.
Place: Memphis, Tenn. Time: Fall, 1930.

Southern Medical Association, Section on Pediatrics

Chairman: Dr. H. Leslie Moore, 1717 Pacific Ave., Dallas, Texas.
Secretary: Dr. Ludo von Meysenbug, 3501 Prytania St., New Orleans, La.
Place: Louisville, Ky.: November, 1930.

STATE

Alabama Pediatric Society

President: Dr. J. MacBell, Mobile.
Secretary-Treasurer: Dr. Hughes Kennedy, Jr., Highland Plaza Apts., Birmingham.

Georgia Pediatric Society

President: Dr. W. N. Adkins, Medical Arts Bldg., Atlanta.
Secretary-Treasurer: Dr. E. N. Gleaton, 212 E. Gaston St., Savannah.

Louisiana State Pediatric Society

President: Dr. J. A. Crawford, Lake Charles.
Secretary-Treasurer: Dr. C. T. Williams, 7401 Burthe St., New Orleans.

Oklahoma State Pediatric Society

President: Dr. William M. Taylor, Osler Bldg., Oklahoma City.
Secretary: Dr. Carol Pounders, 110 W. 10th St., Oklahoma City.
Place: Shawnee. Time: May, 1930.

South Carolina Pediatric Society

President: Dr. M. W. Beach, 126 Meeting St., Charleston.
Secretary-Treasurer: Dr. Julian P. Price, The McLeod Infirmary, Florence.

Texas Pediatric Society

President: Dr. H. P. Ledford, 1310 9th St., Wichita.
Secretary-Treasurer: Dr. John G. Young, 3930 McKinney Ave., Dallas.

Virginia Pediatric Society

President: Dr. Franklin D. Wilson, 136 York St., Norfolk.
Secretary: Dr. J. B. Stone, 2042 Park Ave., Richmond.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. John Toomey, Cleveland City Hospital, Cleveland.
Secretary: Dr. A. B. Grossman, 7016 Euclid Ave., Cleveland.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. G. A. Davis, 12 Belsize Drive, Toronto.
Secretary: Dr. Gladys Boyd, 56 DeLisle St., Toronto.
Time: November 17, December 15, February 16 and March 15.

Bronx Pediatric Society

President: Dr. David I. Frey, 2065 Grand Concourse, New York.
Secretary: Dr. H. M. Eisenoff, 1018 E. 163rd St., New York.
Place: Concourse Plaza, 161st St. and Grand Concourse. Time: Second Wednesday of each month, except June, July, August and September.

Brooklyn Pediatric Society

President: Dr. Joseph F. Paulonis, 1357 Bushwick Ave., Brooklyn.
Secretary: Dr. Irwin Schiff, 658 Eastern Parkway, Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

Central New York Pediatric Club

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Fourth Thursday of October, January and April.

Chicago Pediatric Society

President: Dr. Arthur H. Parmelee, 310 S. Michigan Blvd., Chicago.
Secretary: Dr. Bert I. Beverly, 130 S. Oak Park Ave., Oak Park, Ill.
Place: City Club. Time: Third Tuesday of each month.

Detroit Pediatric Society

President: Dr. Wyman C. C. Cole, 2970 W. Grand Blvd., Detroit.
Secretary: Dr. Charles A. Wilson, 71 E. Perry St., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. Lee Bivings, Exchange Bldg., Atlanta.
Secretary: Dr. William Kiser, Medical Arts Bldg., Atlanta.
Place: Academy of Medicine, 32 Howard St. Time: Second Thursday of each month from October to April, 8 p. m.

Memphis Pediatric Society

President: Dr. G. J. Levy, 899 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. W. R. Graves, 1024 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

Milwaukee Pediatric Society

President: Dr. M. G. Peterman, 167 17th St., Milwaukee.
Secretary: Dr. F. R. Janney, 418 E. North Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

Nebraska Pediatric Society

President: Dr. H. B. Hamilton, 434 Aquila Court Bldg., Omaha.
Secretary: Dr. George L. Clark, 1817 Vinton St., Omaha.
Place: As announced by committee. Time: Second Thursday of each month from October to June, inclusive, 6 p. m.

New England Pediatric Society

President: Dr. Grover F. Powers, New Haven, Conn.
Secretary: Dr. Gerald Hoeffel, 319 Longwood Ave., Boston.
Place: Boston Medical Library. Time: October, January and March.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. F. Elmer Johnson, 300 West End Ave., New York.
Secretary: Dr. Hugh Chaplin, 39 E. 75th St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

North Pacific Pediatric Society

President: Dr. W. F. Patrick, Medical Arts Bldg., Portland, Ore.
Secretary: Dr. L. Howard Smith, Medical Arts Bldg., Portland, Ore.

Northern California Pediatric Society

President: Dr. Edward B. Shaw, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Lloyd Hardgrave, 384 Post St., San Francisco.
Place: Various teaching schools. Time: Monthly.

Northwestern Pediatric Society

President: Dr. E. S. Platou, 730 La Salle Bldg., Minneapolis.
Secretary: Dr. F. G. Hedenstrom, 218 Lowry Bldg., St. Paul.
Place: University Campus and Rochester, Minn. Time: Winter, Spring, Summer, Fall.

Philadelphia Pediatric Society

President: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Secretary: Dr. John D. Donnelly, 115 Bryn Mawr Ave., Bala-Cynwyd, Pa.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

Pittsburgh Pediatric Society

President: Dr. David H. Boyd, 200, 9th St., Pittsburgh.
Secretary: Dr. Theodore C. Elterich, Highland Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate months.

Richmond Pediatric Society

President: Dr. Basil B. Jones, 2618 Grove Ave., Richmond, Va.
Secretary-Treasurer: Dr. John S. Weitzel, 3103 W. Franklin St., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St. Time: First Thursday of each month (other than during the summer).

Rochester Pediatric Society

President: Dr. John Aikman, 184 Alexander St., Rochester, N. Y.
Secretary: Dr. Howard F. Rowley, 278 Alexander St., Rochester, N. Y.
Place: Arranged by program committee. Time: First Friday of each month from October to May.

Rocky Mountain Pediatric Society

President: Dr. Elsie Seelye Pratt, Republic Bldg., Denver.
Secretary-Treasurer: Dr. Wilford W. Barber, Metropolitan Bldg., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month from September to May, inclusive, 2:30 p. m.

St. Louis Pediatric Society

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary-Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

Seattle Pediatric Society

President: Dr. Vernon W. Spickard, Cobb Bldg., Seattle.
Secretary: Dr. Raymond H. Somers, Cobb Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, 7 p. m.

Southwestern Pediatric Society

President: Dr. Ezra S. Fish, Pacific Mutual Bldg., Los Angeles.
Secretary: Dr. Fred L. Glascock, 1136 W. 6th St., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. W. D. Lyon, 101 S. Balch St., Akron, Ohio.
Secretary: Dr. John P. Parsons, 320 S. Division St., Ann Arbor, Mich.
Place: Ann Arbor, Mich. Time: About July 15, 1930.

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